MAJOR RESEARCH PROJECT

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AN INVESTIGATION INTO THE EFFECT OF CAUSAL BELIEFS ABOUT DEPRESSION ON ATTITUDES AND CLINICAL JUDGEMENTS.

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Summary of portfolio

Section A gives an overview of the endorsement, and impact, of biomedical explanations of depression. The review found a weak endorsement of biomedical causal beliefs among lay populations. Professionals were more likely to endorse biomedical and causes. Biomedical beliefs were associated with increased medical help-seeking and reduced blame. They were also shown to increase stigmatising attitudes and reduce preference for psychosocial interventions. The review highlighted a lack of research examining professional attitudes. Clinical and research implications are given.

Section B describes an experimental study into the effect of labelling depression as biological versus psychosocial on clinical judgements and attitudes. Data was analysed using ANOVA. There was small effect of labelling the depression as biological on causal beliefs and judgements of treatment effectiveness. Observational analysis showed that trainees' causal beliefs about the depression had a large effect on judgements. Biological causal beliefs were associated with increased judgements of effectiveness for medication, ECT and hospitalisation and lower perceptions of engagement in psychological therapy. Findings and limitations are discussed within a research and clinical context.

Section C provides a critical and reflective appraisal of the empirical study and the candidates learning and development throughout the process of the research. Future research ideas and clinical implications of the project are discussed.

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Major Research Project

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Section A: Literature Review

Endorsement of biomedical causal explanations for depression: Impact on treatment preferences, help-seeking and stigma.

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Abstract

Introduction.

Biomedical models of mental illness (MI) abound. These have been proposed as helpful in reducing stigma and increasing help-seeking. Research suggests that the consequences of biomedical models of MI are not uniformly positive. Context is given regarding the endorsement and implications of biomedical models of MI amongst lay and professional populations.

Aims.

The review explored the endorsement of biomedical causal beliefs for depression, in lay and professional populations, and the impact of these beliefs on stigma, treatment preferences and help-seeking.

Methods.

Five electronic databases were searched for relevant peer-reviewed articles using keywords. Articles were included if they measured participants biomedical causal beliefs about depression. Twenty-four studies were identified (1991-2011).

Findings.

Although findings were mixed, the review suggests a strong endorsement of psychosocial causal beliefs among lay populations. Professionals and people with severe depression were more likely to endorse biomedical causes. Biomedical beliefs were associated with increased medical help-seeking and reduced blame for depression. Biomedical beliefs were also associated with stigmatising attitudes and reduced preference for psychosocial and self-initiated interventions. Methodological rigour and conclusiveness of findings varied across studies. There was a paucity of studies examining professional attitudes. Clinical and research implications are discussed.

Introduction

Biomedical explanations of MI

Biomedical models were first popularised in the late 19th Century following the discovery that syphilis can cause psychotic symptoms and that certain traits and syndromes are heritable (Hinshaw & Cicchetti, 2000). Their popularity faded following their association with eugenic movements in Nazi Germany (Barondes, 1998). The advent of psychotropic drugs in the 1950's and 1960's led to a resurgence of biomedical theories of MI, such as the serotonin-inactivity model of depression (Borup**,** Meidahl, Petersen & Yangtorp, 1982). Since the 1990's, increased understanding of the brain and genetics has led to a renewed optimism for finding a biological basis for MI and a medical cure. More than 20 years on, there is still little evidence for a genetic or biochemical cause (Hindmarch, 2001; Double, 2004; France, Lysaker & Robinson, 2007).

Integrated 'biopsychosocial' models of MI have been widely adopted within clinical psychology (Read, 2005). These models have been criticised for reducing psychosocial factors to mere triggers of exaggerated genetic predispositions (Read, 2005; Joseph, 2006).

Endorsement of biomedical explanations of MI

In a large review of studies, Angermeyer & Matschinger (1999) found that lay people tend to view MI from a wholly psychosocial perspective. This has been a consistent finding (van Dorn, Swanson, Elbogen &Schwartz, 2005; Read, Haslem, Sayce & Davies, 2006). Mental health professionals have been shown to put more emphasis on biomedical causes of MI than the public (van Dorn, Swanson, Elbogen &Swartz, 2005; Read, Haslem, Sayce and Davies, 2006). Public endorsement of biomedical explanations appears to be increasing. Angermeyer & Matschinger (2005) found that, from 1990-

2001, there was a 19% rise in the endorsement of "brain disease" and "genetics" as causes of MI.

Understanding causal beliefs in MI – Theory

Research has suggested that having an explanation for behaviour encourages the perception of the behaviour as normal – "the understanding it makes it normal effect" (Meehl, 1973). In an experimental study, Kim & LoSavio (2000) found that people judged MIs as more normal and common if a causal explanation was given. They suggested that the effect operates in a similar way to the "simulation heuristic" (Kahneman &Tversky, 1982); which stipulates that an event is judged as more likely to occur if a causal scenario can be easily constructed.

Attribution theory is a framework for understanding the impact of causal attributions in MI (Weiner, 1983; 1985; 1995). Attribution research suggests that the stability and controllability of events affect causal attributions (Weiner, 1983; 1995). Perceiving negative experiences as uncontrollable reduces personal responsibility and maintains self-esteem (Wiener, 1983). Biomedical explanations for MI have been associated with lower perceived control and reduced perceptions of blame and responsibility (Schmidt & Weiner, 1988; Rush, 1998). Medical illnesses may also be perceived to be more stable leading to reduced hope for full recovery (Anthony, 1994). Seligman (1975) proposed that holding a pessimistic attributional style, where negative events are viewed as stable and uncontrollable, can be causal in depression through a process of "learned helplessness."

Clinical Implications - Treatment

Clinicians predict the effectiveness of treatment interventions using formulations of the causal and maintaining factors in a client's distress (Sloman, 2005). Treatments which match causal explanations are judged to be more effective, as are treatments

which act on initial causes rather than symptoms or effects (Yopchick & Kim, 2009). DeKwaadsteniet, Hagmayer, Krol & Witteman (2010) found that professional causal beliefs can lead to bias in the choice of interventions and a disregard of the evidence-base. Strongly endorsing biological attributions has been shown to predict professional preference for pharmacotherapy, hospitalisation and ECT (Read & Harre, 2001).

Lay causal theories also predict treatment preference, with people tending to seek treatments which are congruent with their causal explanations (Read et al., 2006; Furnham, 1991). Congruence may be important in treatment outcomes; clients who hold biological causal beliefs have demonstrated worse outcomes in psychological interventions (Cottraux, Messy, Marks & Bouvard, 1993; Lax, Basoglu and Marks, 1992). Fisher and Farina (1979) found that biomedical beliefs about MI increase professional help-seeking whereas psychosocial models increase self-management.

Jorm et al. (1997) found significant discrepancies between public and professional perceptions of intervention helpfulness. Professionals viewed medical treatments as more helpful than the public, and stress-management, yoga and relaxation as less helpful. Discrepancy between professional and client belief's has been shown to predict worse outcomes and reduced motivation for treatment (Buetler & Clarkin, 1990; Propst, 1980).

Implications - Stigma

Stigma is a process by which a set of people are labelled as different and are stereotyped, disempowered and discriminated against (Link and Phelan, 2001). In a review of studies, Rusch, Angermeyer & Corrigan (2005) found high levels of MI stigma; people with schizophrenia were frequently viewed as dangerous and unpredictable and people with depression were often viewed as weak and incompetent. They found self-stigma was common in MI and included perceptions of personal responsibility,

weakness and low-self efficacy. Self-stigma was associated with a failure to seek help. Stigma is also a significant problem amongst mental health professionals (Gray & Gray, 2002; Schulze, 2007). In a review of studies, Schulze (2007) found mental health professionals attitudes to be the same as, or more negative than, public attitudes.

Studies examining stigma in MI have found a relationship between biomedical beliefs and negative attitudes such as "dangerousness" and "unpredictability" (Read and Law, 1999; Read & Harre, 2001, Walker & Read, 2002), "social distance" (Golding, Becker, Sherman, Rappaport, 1975) and "irresponsibility" (Schwartz & Schwartz, 1977). Illness beliefs are also associated with self-stigma leading people to see themselves as "alien" and less acceptable as a friend (Farina, Fisher, Getter & Fischer, 1978). Professionals endorsing a biological perspective have been shown to view the client as more pathological (Kent & Read, 1998). In an experimental study, Lam, Salkovskis & Warwick (2005) found that psychological explanations reduced judgements of risk, disability and severity of MI. However, the effects of biomedical explanations are not uniformly negative; they have been associated with feelings of self-respect, better self-esteem and lower perceptions of blame (Farina et al., 1978; Read et al., 2006).

Anti-stigma campaigns. Education is a key strategy for reducing stigma (Mayville & Penn, 1998). In 2002, the National Alliance of Mental Illness promoted the message: "mental illness is a brain disease" in an American anti-stigma campaign (Watson, & Corrigan, 2005). Attribution theory has been used to argue that such "illness like any other" approaches will improve attitudes by reducing perceptions of control, responsibility and blame (Read et al., 2006). In the UK, biochemical and illness models of MI are also popular methods in reducing stigma ("Defeat Depression", 1992-1996; "Changing Minds Campaign", 1998-2003; "Time to Change", 2008-2013). Positive outcomes from such campaigns have been limited (Rusch et al., 2005). Mehta & Farina,

(1997) argued that medical illness analogies, used in such campaigns, increase stigma as they encourage perceptions of people with MI as "physically distinct."

Biomedical models and diagnostic labelling

Diagnostic labelling is a key outcome of the biomedical model of mental health used within psychiatry and general practice. The Diagnostic and Statistical Manual allows mental health diagnoses to be made on the basis of clusters of "symptoms" which assume an underlying disease or illness (American Psychiatric Association, 2000). It has been argued that diagnostic labels are an efficient way to distinguish clients based on identifiable characteristics, which enable inferences to be made regarding causal aetiology and in turn allows the effectiveness of interventions to be predicted (Corrigan & Penn, 1999).

A number of critics have argued that the absence of biological or physical markers for mental health diagnoses such as depression, make the biomedical notion of mental illness confused or mythological (Boyle, 1990; Pilgrim & Bentall, 1999). The disease model of diagnosis has also been contradicted by a lack of evidence for effective and specific medical treatments (Moncrieff & Cohen, 2005). These critics argue that depression is a scientifically invalid construct which enables a stigmatising process of labelling and medicalising normal human experience. Nonetheless, the use of diagnostic labels has also become an increasingly prominent feature of psychological services (Boyle, 2007). It has been suggested that the increasing use of diagnosis has led to psychological models of emotional distress being increasingly biomedical or illness based (Read, 2005).

Diagnostic labelling has been criticised due to its implications for stigma. Goffman (1963) described stigma as a social process in which individuals are labelled

with an attribute which is discrediting or shameful. In the context of diagnosis, stigma occurs when the person is labelled as "ill" and is perceived to be different from the norm in a socially significant way. The process of diagnostic labelling involves a subjective value-judgement by mental health professionals, who hold the power to make diagnosis and clinical decisions. The process of labelling and clinical decision-making is also influenced by organisational factors, such as the current introduction of mental health clustering and payment by results within the National Health Service in the UK. In turn, these organisational features are influenced by societal and political factors such as economic pressures or marketing by drug companies. Thus, in considering aetiological influences on clinical-judgements, the wider political and systemic landscape needs to be held in mind.

Review

Aims

The current review explores the impact of biomedical causal beliefs on public and professional attitudes towards depression and its treatment. As previously discussed, the concept of diagnosis and the construct of mental illnesses, such as depression, have been contested. However, the notion that depression is a diagnosable illness is common and the current review aims to explore the implications of endorsing a biomedical or illness explanation of depression. Depression is the most widely diagnosed MI (Murray & Lopez, 1996). Stigma is a significant problem in depression and is associated with not seeking help (Schomerus, Matschinger, & Angermeyer, 2009). Beliefs about causes of depression are broad and include: interpersonal causes (e.g. relationship difficulties), environmental causes (e.g. stress), developmental causes (e.g. childhood abuse),

intrapersonal factors (e.g. attributional style), religious/spiritual causes (e.g. God's will) and biomedical causes (e.g. chemical-imbalance; Addis, Traux, & Jacobson, 1995).

The review aims to answer the following questions:

a.) To what extent do the general public and professionals endorse biomedical/illness explanations as a cause of depression?

b.) Do biomedical causal beliefs impact attitudes towards treatment and help-seeking?

c.) Do biomedical causal beliefs impact stigma?

Methods

Detailed information regarding search methodology can be found in the appendix (A, p.102). Peer-reviewed articles were included if they measured biomedical causes of depression and addressed any of the review aims. Five electronic databases: PsycINFO, Ovid MEDLINE, Cochrane library, Web of Science and ESBCOhost, were searched using the following keywords (plus synonyms): "biological beliefs" OR "biomedical beliefs'' OR "illness beliefs'' AND "depression" AND/OR "attitudes" OR "stigma" OR "treatment preference" OR "help-seeking"

Twenty-four relevant studies were identified (published 1991-2011). For a description of the studies see Appendix (B, p.103), and see Table 7(Appendix C, p.104) for an outline of the main findings,).

Large-Scale Surveys

Lay populations. McKeon and Carrick (1991) found two-thirds of the people they interviewed did not perceive depression to be an illness. Causal beliefs about depression reflected a psychosocial understanding, with stress and bereavement being most commonly cited. Only 9% mentioned "chemical-imbalance" as a causal factor.

Participants expressed few negative attitudes towards depression and 73% felt it could be successfully treated. The authors conclude that the lack of illness explanations for depression explains the low prevalence of stigmatising attitudes. This conclusion goes beyond the data in their study as they did not find any significant association, or causal link, between illness beliefs and stigmatising attitudes.

Jorm et al., (1997) assessed beliefs about depression and schizophrenia through the use of case vignettes. The most common causal explanations for both vignettes were day-to-day stressors, trauma and bereavement. Half of the sample endorsed "genetics" as being causal in depression and the same number endorsed "weakness of character". Participants who correctly recognised the vignette as depression made more social attributions and were less likely to endorse "weakness of character". The findings suggest that holding a social understanding of depression is associated with better recognition and a less negative evaluation of the sufferer. However, the authors did not directly test this association.

Paykel, Hart & Priest (1998) evaluated the "Defeat Depression" campaign (UK: 1991-1996) which utilised an illness approach. The authors compared household attitude surveys from 1991 and 1997. The authors conclude that between these time-points illness beliefs became more common therefore demonstrating an increase in favourable attitudes to depression. Yet, it is not clear that viewing depression as an illness is synonymous with favourable attitudes. Examining the survey results shows there was no change in the percentage of people who viewed people with depression as "mad/unstable" and there was a decrease in the percentage who thought depressed people "deserve more support from family and friends." There was a 10% increase in the number of people endorsing "biological changes in the brain" (43% in 1997). Endorsement of anti-depressant treatment also increased significantly over the time

period. Willingness to seek professional help increased by 8% from 1991-1997. However, there was no reduction in the number of people who would feel embarrassed to see a GP (62%).

The evaluation is limited by the number of multiple comparisons made without adjustment of the significance level. In addition, "don't know" answers were collapsed into the "disagree" category. This may have inflated the percentage change in positive attitudes; people who answered don't know to statements such as "depressed people are mad/unstable" would have been counted as having a favourable attitude. This is particularly problematic in the context of social desirability in face-to-face interviews. Although the campaign led to increased medical help-seeking, it does not appear that the "illness-like-any-other" approach was effective in reducing stigma.

Lauber, Falcato, Nordt & Rossler (2003) conducted a telephone survey in which they asked people to generate possible causes of depression for a case vignette. Psychosocial explanations were the most commonly cited. Illness explanations were also common in this sample and cited by 25%. In contrast to Jorm et al., (1997), recognition of the vignette as depression correlated with higher ratings of heredity and illness explanations. This difference may reflect an increase in illness beliefs over time.

Jorm and Griffiths (2008) conducted a survey exploring stigma towards depression and schizophrenia. Stigma was conceptualised as "dangerousness" and "social distance." They found a link between biomedical explanations and stigma for schizophrenia but not for depression. However, dangerousness may be less relevant in depression stigma (Rusch et al., 2005). When vignettes were labelled as "depression," there was a trend towards biomedical explanations leading to a greater desire for social distance compared to psychosocial explanations.

Pescosolido et al. (2010) compared attitudes to MI on the 1996 and 2006 American General Social Survey. Participants were randomly assigned one of three vignettes (schizophrenia, depression or alcohol dependence). They found that significantly more people endorsed biological explanations of all three vignettes in 2006. For depression, there was a 13% rise in chemical-imbalance explanations and a 12% increase in genetic explanations. Seventy-two per cent of participants in 2006 viewed depression as an illness compared to 65% in 1996. Participants were 10% more likely to state the depressed individual needed to see a psychiatrist and 9% more likely to state they need medication. Stigma remained high across time-points. Biomedical attitudes were found to be either unrelated to stigma, or to lead to an increase in negative attitudes. Endorsing biomedical explanations for depression was associated with viewing the depressed person as more dangerous and an increased desire for social distance.

The 1990's in American was designated the "decade of the brain" based on the assumption that advances in neuroscience would hold the key to understanding mental illnesses and reducing stigma (Jones & Mendell, 1999). The results of this study suggest that although endorsement of biological explanations and medical treatment for depression increased significantly in America between 1996 and 2006, stigmatising attitudes remained fixed.

Depressed populations. In a UK survey, Ogden et al. (1999) found that GPs equally endorsed both biomedical and psychological causes of depression whereas patients more frequently endorsed psychosocial reasons. Patients who had experienced depression had similar beliefs to GPs. This suggests that people who have been depressed view biomedical causes as more relevant in their explanation for depression. The authors do not investigate this finding and there are a number of

possible explanations. Firstly, people who experience depression may have a more pessimistic attributional style which is congruent with biomedical explanations. Secondly, having depression leads to a shift in causal attributions to reduce feelings of blame. Thirdly, GPs provide psycho-education to patients which encourages a biomedical conceptualisation of symptoms. Finally, pharmacological treatment may lead to a shift in causal beliefs to promote congruency.

Budd, James & Hughes (2008) surveyed service-users' explanations for their depression and perceived helpfulness of interventions. The most important causes were: bereavement, biochemical-imbalance, and sexual abuse. Stronger endorsement of a biochemical-imbalance explanation of depression was associated with greater perceived helpfulness of medication. There were no other associations between treatment beliefs and causal explanations. This study suggests that biochemical explanations, in combination with personal trauma, are important in service-users' explanations for depression and preference for anti-depressant treatment. The findings of this study may not generalise to the wider population of people experiencing depression, as these participants had long histories of depression (average of 17 years) and had attempted multiple treatments.

Hansson, Chotai & Bodlund (2010) surveyed patients with depression from 46 health centres across Sweden. The most commonly cited causes for depression were stress and personality. Biological causes were cited by just 3.6%. Despite the low endorsement of biological causes 83% of participants were prescribed anti-depressants compared to 9% receiving psychotherapy.

Single-Site Surveys and Observational Studies

Lay populations. In contrast to previous studies, Goldstein & Rosselli (2003) found an association between biological beliefs and more positive attitudes to

depression, in a sample of American students. Factor analysis of generated causes for depression demonstrated three models of aetiology representing biological, environmental and psychological beliefs, with the biological factor being most strongly endorsed. Holding a biological model of depression was associated with reduced perceptions of blame, more positive beliefs towards depression, greater help-seeking and greater perceived effectiveness of psychotherapy. In contrast, psychological models were associated with increased blame and desire for social distance.

France, Lysaker & Robinson (2007) also found that American undergraduates strongly endorse biomedical causes of depression. In a free response task, chemical-imbalance was the most commonly cited cause of depression. Over half the sample rated chemical-imbalance as the primary cause of depression. Those endorsing the chemical-imbalance explanation were more likely to seek-help help from a range of mental health professionals. The high endorsement of chemical-imbalance explanations in American samples may reflect the high prevalence of direct-to-consumer advertising for anti-depressants and the resulting exposure to the chemical-imbalance hypothesis (Hinshaw, 2006).

In a similar study, Nieuwsma & Pepper (2010) explored the impact of etiological beliefs on stigma, self-efficacy, and treatment effectiveness in American undergraduates. The most important causal factors in depression were rated as negative life events, recent misfortunes and a chemical-imbalance. People who endorsed psychosocial explanations were more likely to view self-initiated treatments as effective. There was a trend towards endorsing a biological explanation of depression and greater perceived effectiveness of medication. No significant association was found between causal explanations and stigma.

Wong, Tran, Kim, Kerne & Calfa (2010) conducted a survey examining Asian-American's attitudes to mental health. Participants were presented with a depression vignette and asked to provide possible labels, causes, consequences, and solutions for the difficulties. Content analysis determined the most commonly cited causes were interpersonal. Biological reasons and contextual causes were associated with professional help-seeking. Logistical regression found that those endorsing biological reasons were 1.65 times more likely to seek professional help.

Depressed populations. Srinivasan, Cohen & Parikh (2003) surveyed psychiatric outpatients. Participants identified stress or negative life events and cognitive style as partial causes for their depression whereas biomedical causes were not endorsed. Women were more likely than men to endorse "biological abnormality" as a cause. The authors argue that low endorsement of biological explanations explains poor compliance with anti-depressant treatment. This study lacks measures of medication compliance and/or treatment preferences which might have provided evidence for the authors' conclusion. In addition, all causal ratings were very low. The few, broad causes of depression used in this study may not have encompassed the many possible explanatory reasons people hold for depression. For instance, "biological abnormality" may, or may not, be viewed to include: chemical-imbalance, hormonal changes, brain damage and physical illness.

Brown et al., (2007) examined the relationship between illness beliefs and functioning in service-users with mild-moderate depression. The most strongly endorsed reason for depression was stress. Regression analysis demonstrated a significant correlation between medical illness beliefs and lower perceptions of control over symptoms and greater perceived consequences. The conclusiveness of the findings is limited by the number of regression analysis conducted without directional

hypothesis or conservative *p* values. Thus the small correlations found in this study appear unremarkable.

Leykin, DeRubeis, Shelton & Amsterdam (2007) explored the impact of treatment (anti-depressant medication; ADM, versus cognitive therapy; CT) on participants' beliefs about the causes of their depression following successful treatment. Participants were part of a RCT evaluating the effectiveness of CT. Data on characterological beliefs (conceptually related to CT e.g. depressive thinking) and biomedical beliefs (e.g. chemical-imbalance) were collected on all participants in active treatment. Characterological reasons were the most strongly endorsed reasons for depression across the sample, followed by childhood events and biological causes. The high endorsement of characterological beliefs in this study contrasts with previous findings, suggesting it may be related the sample recruited for the RCT. Causal beliefs were not significantly associated with treatment outcome although the authors report a trend towards higher biological beliefs and worse outcomes in CT. Participants successfully treated with CT reported weaker biomedical beliefs post-treatment, whereas participants successfully treated with ADM reported weaker characterological beliefs. The results suggest that successful treatment confirms treatment congruent beliefs and "weeds out" treatment incongruent beliefs.

Meyer & Garcia-Roberts (2007) examined how congruence between reason-giving for depression and treatment-type impacts motivation in patients receiving psychological therapy in the UK. Cognitive reasons were the most commonly endorsed cause for depression. Biological reason-giving varied significantly as a function of depression severity with the most severely depressed endorsing biological causes more than those with mild-moderate depression. Congruence between reasons for depression and interventions increased treatment motivation. People with severe

depression were more motivated for biological treatment and less motivated for interventions targeting childhood issues. This supports previous findings that people with severe depression are more likely to hold biological explanations for depression and to be more motivated for biomedical treatments. The reason for this is not clear; it seems feasible that people with severe depression may perceive their symptoms to be more stable leading to low self-efficacy. A limitation of this study is that all participants were receiving psychological therapy, primarily CBT. This bias may explain the high endorsement of cognitive reasons amongst participants.

In a Deutsch study, Schweizer et al. (2010) examined the impact of causal explanations on treatment preferences in a people diagnosed with major depression. Treatment options were: CBT, Interpersonal Therapy (IPT) or pharmacotherapy (PHT; pure or combined with therapy). Biological reasons were the least cited cause of depression and pure PHT was the least popular treatment. Intraindividual attributions were associated with a preference for CBT and biological attributions were associated with a preference for PHT. Experience of "failed" treatment attempts was associated with higher endorsement of biological explanations of depression. In the context of previous research it seems plausible that biological explanations help to reduce perceived responsibility for "failing" treatment.

In a similar study, Khasla, McCarthy, Sharpless, Barrett and Barber (2011) examined the link between causes for depression and treatment choice in participants with major depression who were recruited for an American RCT. "Childhood" and "characterological" were the most cited causes of depression, followed by "biological" causes. Participants from ethnic minorities were less likely to endorse biological explanations than Caucasians. Regression analysis demonstrated an association between receiving previous treatment and higher endorsement of biological

attributions. There was a trend towards strongly endorsing the biochemical-imbalance explanation of depression and a preference for medication. Those who preferred psychotherapy were more likely to endorse childhood reasons as the cause of their depression. Forty-one per-cent of participants preferred anti-depressant treatment to psychotherapy, suggesting that for many people preference for medication was unrelated to causal attributions.

Professionals. Kuyken, Brewin, Power & Furnham (1992) compared causal beliefs about depression across clinical psychologists (CPs), depressed service-users (SUs), and lay people in Greater London. There was a significant difference in the frequency of biomedical responses between the three groups; 65% of SUs cited biomedical causes as a reason for depression compared to 48% of CPs and 14% of lay people. CPs and SUs rated drug treatment as more effective than did lay people. SUs and CPs viewed drug treatments to be as effective as psychotherapeutic and social interventions. In contrast, lay people gave far lower efficacy ratings for medical treatment than for all other interventions.

Quasi-Experimental and Experimental studies

Lay populations. Han, Chen, Hwang & Wei (2006) used an experimental design to evaluate the effectiveness of different educational messages on willingness to seek-help amongst undergraduates in Taiwan. Students were randomly assigned to four groups: biological education; de-stigmatisation education; combined; or a no education control group. Participants in the experimental groups were asked to read a paragraph on depression along with information on its biological aetiology and/or educational material aimed at reducing perceived blame. The authors developed three questionnaires to examine biological attribution, blame and help-seeking willingness.

Biological education significantly increased willingness to seek help. The de-stigmatisation education reduced personal blame but did not influence help-seeking.

There are a number of limitations of this study. Firstly, help-seeking was measured primarily by examining attitudes to seeking help from medical professionals. It seems logical that participants who attribute the cause of depression to biomedical causes would seek medical help for a solution. Other studies suggest that biological attribution also results in reduced self-efficacy and less motivation for self-initiated interventions (Nieuwsma & Pepper, 2010). Thus the authors' conclusion that promoting biomedical explanations for depression is a positive strategy for public education, based on a single measure of help-seeking, appears flawed.

Rusch, Kanter and Brondino (2009) examined the impact of causal information about depression on the effects of stigma-reduction video-messages. Stigmatising attitudes and behavioural intentions to disclose the depression were measured pre- and post-test and at one-week and one-month follow-up. Participants were randomised to either of the two experimental conditions ("contextual" versus "biological" causes) or either of an active control (programme containing no causal information) or non-active control (no programme).The contextual and control programmes both had a large effect on stigma-reduction. The biomedical programme did not significantly reduce stigma. Having causal beliefs about depression which were congruent with the programme content significantly increased their effectiveness. People in the contextual condition reported higher behavioural intentions than those who watched the biological or contextual programmes. In line with theory (Meehl, 1973), findings from this study suggest that having an explanation (which is non-stigmatising) is important in disclosing depression to others and seeking social support. Unfortunately this study did not explore willingness to seek professional help.

Deacon and Baird (2009) recruited psychology undergraduates at an American University. Participants were presented with either a chemical-imbalance or a biopsychosocial explanation of depression. Participants were asked to complete attitude questionnaires as if they had received a diagnosis of depression. Participants were then given the alternative explanation for depression and the questionnaires were repeated. The biochemical explanation was rated as the most credible and was associated with lower ratings of responsibility for the depression. The biochemical explanation was also associated with significantly worse ratings of prognosis and psychotherapy-efficacy. The biochemical explanation was associated with greater perceived effectiveness of medication whereas the biopsychosocial explanation was associated with greater effectiveness of self-initiated interventions. All effect sizes were moderate to large. The authors conclude that the chemical-imbalance explanation for depression leads to less personal responsibility and blame but also leads to a worse expected prognosis and an expectation that psychosocial treatment will be largely ineffective. Extrapolating these findings to a wider population needs to be done with caution. The repeated measures design used in this study may have led participants to present more polarised views thereby increasing effect sizes.

Professionals. Ahn, Proctor and Flannagan (2009) conducted three studies with mental health professionals and trainees in America. In study one, participants were asked to rate the extent to which each of 445 disorders were biological, psychological or environmental in nature. A strong negative correlation was found between biological and psychological causal ratings, whereas there was a strong positive correlation between ratings of psychological and environmental causes. Both psychologists and psychiatrists gave significantly higher ratings of biological causation across the disorders than social workers. The pattern of correlations supported a continuum of

etiological beliefs from disorders viewed as primarily biological (e.g. autism) to disorders seen as primarily psychosocial (e.g. bulimia). Depression fell in the middle of this spectrum and was rated as both moderately biological and psychosocial. The second study replicated these findings in a sample of 63 registered clinicians.

In their final study, Ahn et al. examined the extent to which causal beliefs impacted clinicians' judgements of treatment efficacy in conditions which had been rated as moderately biological. Participants were given patient vignettes in which each patient was described as having a MI with a specific cause (e.g. depression caused by genetics). Repeated-measures ANOVA found a significant interaction between cause-type (biological vs. psychological) and treatment-type (medication vs. therapy). When participants were told the cause of depression was biological they rated medication as more effective than therapy, whereas participants who were told the cause was psychological rated therapy as more effective. The pattern of results was independent of profession. The authors argue that a focus on causal explanations might blind clinicians to the benefit of different treatment approaches.

Discussion

a.) To what extend do the general public and professionals endorse biomedical/illness explanations as a cause of depression?

The studies reviewed suggest that lay causal beliefs about depression tend to reflect a psychosocial framework. Eight of 12 studies cited psychosocial causes as the most commonly endorsed explanations, including all four representative population surveys. Two longitudinal surveys found significant increases in biomedical beliefs over time in the UK (Paykel et al, 1998) and America (Pescosolido et al., 2010). Four small-scale surveys found strong endorsement of biomedical beliefs. These studies had

unrepresentative samples (American psychology undergraduates). The contrasting findings may also represent more exposure to biomedical theories of depression in American culture (Hinshaw, 2006). Findings suggest that people from non-Western cultures are less likely to adopt biomedical explanations for depression (Wong et al, 2010; Khasla et al. 2011). Seven of 10 studies, found people with experience of depression strongly endorse psychosocial causes. Biomedical causal beliefs were more common for people with severe depression (Budd et al., 2008; Meyer & Garcier-Roberts, 2007). Three studies examined professional beliefs (Ahn et al., 2009; Ogden et al., 1999; Kuyken et al., 1992). These suggest that professionals put equal emphasis on both biomedical and psychosocial causes in depression.

Limitations. Measurement reliability of causal beliefs varied across the studies. The majority of studies asked people to rate the likelihood of possible causes. Few studies asked participants to rank the relative importance of these causes. Although some studies utilised validated scales of aetiological beliefs many used self-designed scales which did not appear to encompass the full range of possible causal beliefs. Few potential moderating variables were measured across any of the studies. It would be helpful to understand how personal characteristics, contact with MI, and education levels influence causal beliefs.

b.) Do biomedical causal beliefs impact attitudes towards treatment and help-seeking?

The studies reviewed suggest that lay endorsement of biomedical beliefs leads to greater professional help-seeking (Paykel et al., 1998; Goldstein&Roselli,2003; Han et al., 2006; Wong et al., 2010); greater perceived effectiveness of medication (Kuyken, 1992; Budd et al., 2008; Deacon & Baird, 2009), a preference for medical treatment

(France et al., 2007; Meyer & Garcia-Roberts, 2007; Schweizer et al., 2010)and less preference for talking therapies (France et al.,2007). In contrast psychosocial beliefs were associated with a preference for psychotherapy (Meyer & Garcia-Roberts, 2007; Schweizer et al., 2010; Khasla et al., 2011) and greater perceived effectiveness of self-initiated interventions (Goldstein & Roselli 2003; Nieuwsma & Pepper, 2010). Ahn et al., (2009) found that giving professionals' biomedical causal information leads to greater perceived effectiveness for medication and lower perceived effectiveness for psychotherapy.

Limitations. The majority of these studies used correlational designs which cannot infer causality. None of the studies examined potential moderating variables.

"Help-seeking" in many of the studies was primarily measured by medical help-seeking and did not explore willingness to seek psychological help or help from religious leaders, friends or family. Using medical help-seeking as a measure of self-stigma, as was the case in many of the studies, did not seem valid.

Only one study examined how service-users' experience of treatment effectiveness impacted their causal explanations of the depression. In addition, it would have been interesting to explore how congruency between service-users' explanations for depression and their treatment intervention impacted adherence, motivation, treatment effectiveness and experiences of the intervention.

c.) Do biomedical/illness causal beliefs impact stigma?

The findings suggest that biomedical causal beliefs impact stigma. Biomedical beliefs were associated with less control over symptoms (Brown et al., 2007; Deacon & Baird, 2009), lower self-efficacy (Deacon & Baird, 2009), less willingness to disclose depression (Rusch et al., 2009), worse prognosis (Deacon & Baird, 2009), increased

desire for social distance (Jorm & Griffiths, 2008) and greater perceptions of dangerousness (Pescosolido et al., 2010). However, biomedical beliefs were also associated with less self-blame and responsibility (Deacon & Baird, 2009; Goldstein & Roselli, 2003). Longitudinal studies suggest that increases in biomedical beliefs were associated with no change in, or a worsening of, attitudes over time (Paykel et al., 1998; Pescosolido et al. 2010).

Limitations. None of these studies employed robust experimental designs so they cannot infer causation. Analyses were often limited by multiple comparisons without the use of a conservative *p* values meaning results at the $p<.05$ level need to be treated with caution. Some of the significant correlations were small and without effect sizes it is unclear how meaningful these findings are.

Few of the studies explored the potential moderating effects of factors such as gender, age or the severity of the depression on aetiological explanations and stigma. Factors which may have moderated stigma, such as social contact, were also not explored. In addition, the longitudinal studies which compared stigmatising attitudes over time did not account for generation effects. Thus the small changes in attitudes reported in these studies may be a result of changes in the cohorts being sampled.

Limitations of the review

There was a paucity of research examining a range of professional attitudes especially in the areas of stigma and judgements of treatment effectiveness. Where studies did explore professional attitudes, many did not explicitly state the professions which were sampled making it difficult to compare the effect of biomedical beliefs on attitudes and clinical judgements across professional groups. Treating "professionals" as a homogenous group may have masked variability in aetiological beliefs, clinical

judgements and stigma across the different professions which are involved in the treatment of people with depression.

A further limitation of the review was the lack of any qualitative studies. Only two thematic analytic studies were identified in the literature review (Gammell & Stoppard, 1999; Schreiber & Hartrick, 2002). These examined Canadian women's conceptualisations of depression as a medical illness. These studies were excluded to aid coherence of the literature review. However, the lack of qualitative studies precluded the exploration of the personal narratives and discourse that surround the construct of depression as a medical illness.

Implications for research and practice

The review suggests that promoting biomedical causes of depression may reduce self-blame at a cost of reduced self-efficacy and perceived control over symptoms. Although, promoting biomedical beliefs encourages medical help-seeking, promoting biomedical models of depression may reduce preference and motivation for self-initiated and psychological treatments. Understanding clients' causal beliefs may be helpful in exploring motivational issues and self-stigma when planning treatment. Psycho-education which emphasises biomedical predispositions may be incongruent with lay models of depression and counter-productive. Biomedical beliefs may also increase stigmatising attitudes to people with depression and lead to a desire for social distance.

Overall, there is little evidence that biomedical models of depression are helpful in reducing stigma. However, in line with previous literature it does appear that having an explanation is important in increasing behavioural intentions such as willingness to disclose depression (Meehl, 1973). The findings suggest that psychosocial or contextual

explanations may be more acceptable and less stigmatising to the public than biomedical explanations.

None of the studies in this review examined the impact of clinicians' causal beliefs on stigma and only three studies examined clinician's treatment preferences. These studies suggest that clinicians' causal beliefs may lead to treatment preferences which could be incongruent with the evidence-base. Further research is needed which examines the impact of causal beliefs on clinicians' attitudes towards depression and treatment. In addition, it would be useful to explore whether clinicians' causal beliefs affect stigmatising attitudes towards the client. This research would benefit from using a robust experimental design to allow for causal inferences to be made.

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Major Research Project

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Section B: Empirical Paper

"Typical of Biological Depression:" Are Clinical Judgements Prone to Causal Bias? A Preliminary Investigation.

Word Count: 8578 (579)

Abstract

Aims. Biological explanations of depression have been found to increase professional perceptions of the effectiveness of medical treatments and reduce the perceptions of the effectiveness of psychological therapy. Studies in lay populations have shown that biological explanations reduce perceptions of self-efficacy and control over depression symptoms. There is a lack of research examining the impact of causal models on clinicians' attitudes. The current study aimed to explore whether clinicians' causal models of a client's depression can be biased by aetiological labelling and, in turn, whether clinicians' causal models impact clinical judgements and attitudes.

Design. An experimental design was utilised, with one independent variable (labelling of the client's depression) with three levels (biological, psychosocial and neutral). Outcomes measured causal beliefs, treatment effectiveness, control, clinical attitudes and perceived stigma in relation to a client vignette. Observational data were analysed to explore the effects of clinicians' primary causal models on the outcome variables.

Methods. Over 200 trainee clinical psychologists, across England, Scotland and Wales, took part in an online survey, presented using surveymonkey®. Where appropriate data were analysed using ANOVA.

Results. There was a small effect of the manipulation; labelling the depression as biological increased biological causal attributions and increased perceptions of the effectiveness of medical treatments. The exploratory analysis demonstrated substantial effects of strongly endorsing biological causal beliefs on judgements of medical treatments and client engagement.

Conclusions. The results suggest that clinicians' casual models of a client's depression may bias clinical judgements. These findings are preliminary and further research is needed.

Introduction

Causal models of depression

A dichotomy has often been cited between biological and reactive types of depression (Beck and Alford, 2009). The former has been attributed primarily to biological causes, such as chemical imbalances and genetics, and the latter to psychosocial reactions to stressors, such as job loss or bereavement. The validity of such a distinction has been questioned and it has been argued that it reflects an unscientific mind-body dualism (Pies, 2009).

Following the advent of psychotropic drugs in the 1960s, there was a surge in biomedical explanations for depression, such as the serotonin-inactivity theory (Borup, Meidahl, Petersen & Yangtorp, 1982). Increased scientific understanding in the 1990's, led to optimism for finding a neurological or genetic basis for depression (Jones & Mendall, 1999). More than 20 years on, there is little evidence for a primary biomedical cause of depression (Double, 2004; Hasler, 2010; Hindmarch, 2001). In parallel, there has been an increase in psychological and psychosocial theories of depression, including models of: cognitive-mediation (Beck, 1964), stress-coping appraisal (Billings & Moos, 1982), learned helplessness (Seligman, 1975) and metacognitive processes (e.g. Sheppard, & Teasdale, 2000). Psychosocial models have been criticised for a lack of empirical evidence as complete models of depression (e.g. Coyne & Gotlib, 1983; Hahner, 1989). Despite these criticisms there continues to be little integration between psychological and biological models of depression (Lam, Salkovskis & Warrick, 2005).

In clinical practice the use of integrated biopsychosocial models have been encouraged to take into account the many causal variables in mental illnesses (Engel, 1977). However, biopsychosocial models have been criticised for reducing psychosocial factors to mere triggers of exaggerated genetic predispositions (Joseph, 2006; Read,

2005). Although many clinicians claim to endorse biopsychosocial models, experimental studies suggest that clinicians tend to attribute the primary cause of mental disorders to either biological or psychosocial causes (Ahn, Proctor & Flanagan, 2009). In addition, clinicians have been found to more strongly endorse biological causes of depression compared to the public (Kuyken, Brewin, Power & Furnham, 1992; Ogden et al., 1999).

Biomedical models and diagnostic labelling

The process of diagnosis is a key outcome of biomedical models of depression. The use of diagnostic labels has become a more prominent feature within mental health and psychological services (Boyle, 2007). The increase in diagnostically driven psychology services has led to authors arguing that psychological models of mental health problems are becoming increasingly biomedical and illness based (Read, 2005). In the face of the dominant medical discourse surrounding experiences of emotional distress within mental health services, there is much debate around the construct of depression and its diagnosis. Depression is the most commonly diagnosed mental health problem (World Health Organisation, 2008). It has been argued that such diagnostic labels are an efficient way to distinguish clients based on identifiable characteristics, thereby enabling inferences to be made regarding aetiology and the effectiveness of interventions (Corrigan & Penn, 1999). On the other hand, critics have proposed that there is an absence of any biological or physical markers for depression, making the diagnosis of a depressive illness confused and erroneous (Boyle, 1990; Pilgrim & Bentall, 1999). Such critics argue that depression is a scientifically invalid construct which enables an unhelpful process of labelling and medicalising human misery.

The role of causal explanations in treatment

Despite the prevalence of the diagnosis of depression, around 50% of people in developed countries do not receive any treatment. A leading cause of not seeking help is social stigma (WHO, 2008). Holding an explanation for depression has been associated with reduced stigma and increased help-seeking (Rusch, Kanter and Brondino, 2009). Meehl (1973) proposed that having causal explanations for experiences such as depression is important in judgements of normality – "the understanding it makes it normal effect." Kim and LoSavio (2000), found that people judged mental illnesses to be more common and more normal if coherent causal explanations were given.

Identifying causal and maintaining factors is a key part of clinical formulations, and is used to predict treatment effectiveness (Sloman, 2005). Yopchick & Kim (2009) found that clinicians judge treatments to be more effective if they are perceived to act on initial causes rather than symptoms.

Biological versus psychosocial causal explanations

Biological and psychosocial causal models may have inadvertent consequences on attitudes and behaviour. Attribution research (Weiner, 1983; 1985; 1995) has found that perceiving negative events as uncontrollable reduces personal responsibility and maintains self-esteem (Weiner, 1983). In depression, it has been proposed that biological causes, such as genetics, are viewed as less under the individual's control, thereby reducing blame and encouraging help-seeking (Deacon & Baird, 2009; Paykel et al., 1998). This idea has led to the promotion of depression as a biological illness in a bid to reduce stigma and encourage help-seeking in depression (e.g. "Defeat Depression," 1992-1996, Paykel et al., 1998). The results of these campaigns have been mixed. Biological explanations have been associated with reduced perceptions of blame

(Brown et al., 2007; Deacon & Baird, 2009) and increased professional help-seeking (Goldstein & Roselli, 2003; Paykel et al., 1998). Biological explanations have also been associated with reduced motivation for psychological therapy, lower self-efficacy and a worse expected prognosis compared to psychosocial explanations (Deacon & Baird, 2009; France et al. 2007; Schweizer et al., 2010). These consequences can be explained by attribution research if biological causes of depression are perceived to be more stable than psychosocial causes. In experimental studies, Weiner (1995) found negative events attributed to a stable cause can reduce self-esteem, self-efficacy and motivation.

The consequences of labelling depression as a biological illness may also have important consequences for stigma. Goffman's (1963) seminal work, explored the concept of stigma as a social process in which an individual is labelled with an attribute which is discrediting and shameful. In the context of depression stigma may occur when the person is labelled as being "ill" and is perceived to be different from the norm.

Causal explanations and clinical judgements

The process of labelling depression as biomedical or psychosocial involves subjective value judgements by those who hold the power to make clinical decisions. In addition, wider systemic factors impact upon decision making in a complex process with multiple individual, organisational, social and political influences. For example, within the NHS there is an increasing use of mental health clustering based largely on medical diagnosis coinciding with economic pressures and payment by results. Thus in considering the impact of aetiological labelling on clinical judgements these wider systemic factors need to be borne in mind.

Research into clinical decision-making has found that clinicians' causal models are strong predictors of treatment strategies (Witteman & Keole, 1999). Furthermore, experimental studies have shown that clinician's own causal beliefs can lead to a bias in

clinical decision-making in which clinicians ignore client-specific data. (DeKwaadsteiniet, Hagmayer, Krol & Witteman, 2010). Research has shown that incongruence between treatment interventions and client's causal beliefs can lead to worse outcomes and reduced motivation for treatment (Cottraux, Messy, Marks, Mollard, & Bouvard, 1993; Lax, Basoglu and Marks, 1992).

Clinical studies have shown that professionals who strongly endorse biological causal explanations are more likely to advocate pharmacotherapy, hospitalisation and ECT (Read & Harre, 2001) and to view the client as more pathological (Kent & Read, 1998). Conversely, Miresco and Kirmayer (2006) found that clinicians feel clients are more responsible, and to blame, for "psychological" symptoms compared to "biological" symptoms.

There is a paucity of research examining the impact of professionals' causal explanations on clinical judgements in depression. In a search of the literature, only two relevant studies were found. Kukyen, Brewin, Power & Furham (1992) found that stronger biomedical causal beliefs among clinical psychologists, relative to the public, were associated with greater perceived effectiveness of medication. In an experimental study, Ahn, Proctor & Flanagan (2009) demonstrated that clinical psychologists' judgements can be biased by causal information. Giving clinicians a biological causal explanation for a client's depression (e.g. genetics) increased ratings of effectiveness for medication and reduced ratings of effectiveness for psychological therapy.

The finding that clinicians' causal models can bias treatment decisions is of particular significance given the prominent role evidence-based guidance in clinical practice. The National Institute of Clinical Excellence's guidelines for depression promote a stepped-care model of treatment, from active-waiting and guided self-help to

high-intensity interventions such as Cognitive-Behavioural Therapy (CBT), Interpersonal Therapy (IPT) and anti-depressants (NICE, 2009).

The NICE guidance for depression states:

> "Do not routinely vary the treatment strategies for depression described in this guideline either by depression subtype or by personal characteristics as there is no convincing evidence to support such action (NICE, 2009, p.28)"

The Present Study

The present study examines: a) whether clinical psychology trainees' causal beliefs can be modified through the presentation of a client's depression as being "biological" or "psychosocial," b) if any modification in causal beliefs affects clinical judgements and attitudes, and c) whether clinicians' primary causal beliefs about the depression bias clinical judgements and attitudes.

"Clinicians", in the context of this study, refers to trainee clinical psychologists. Previous research suggests that clinical psychologists are as likely to be biased by aetiological information in judging the effectiveness of treatments for depression as medical doctors or social workers (Ahn et al., 2009). Investigating the effect of causal labelling on clinical judgements is of particular interest within clinical psychology due to the central role of formulation in clinical practice. Clinical psychologists often draw upon multiple theoretical models in formulating the causal and maintaining factors in a client's distress and use these formulations to plan treatment. If causal attributions affect clinicians' preferences and optimism for treatment, understanding this will be important in considering the role of clinicians' causal explanations in clinical practice. For example, will a clinician who perceives the cause of depression to be more biological perceive psychological therapy to be less effective? Ultimately, clinicians'

judgements and optimism for treatment are likely to affect the client relationship and treatment outcomes. Also of significance, is whether causal bias can lead clinicians to make judgements which are not justified by the best available evidence.

Hypotheses

Clinical psychology trainees presented with a client vignette in which symptoms are proposed to be typical of a "biological depression" relative to trainees presented with a vignette in which symptoms are proposed to be typical of a "psychosocial depression" will:

1.) Be more likely to attribute the cause of the clients' depression to biological factors.

2.) Be more likely to advocate medical treatment for the depression and be less likely to advocate psychological or self-initiated therapies.

3.) View the client as being less self-efficacious and the depression as being less controllable.

4.) Demonstrate more negative attitudes towards the depression and more pessimistic attitudes towards treating the client psychologically.

5.) Have different perceptions of the likelihood of the client experiencing stigma.

Method

Design

The study implemented an experimental design, with one independent variable (aetiology) with three levels: biological, psychosocial and neutral (control). Participants were randomly allocated to conditions and were blind to the manipulation. Questionnaire data was collected on six outcomes: causal beliefs, treatment effectiveness, control, self-efficacy, perceived stigma, and clinicians' attitudes. Data was analysed using SPSS version 20.

Participants

Power analysis was conducted using g*power (Faul, Erdfelder, Lang, & Buchner, 2007). To demonstrate a medium effect (d= 0.5), in a one-tailed test (p= .05), at 1–β= 0.86, a minimum of 41 participants were needed per group. To allow for data to be analysed non-parametrically, this calculation was multiplied by 1.05 to give a minimum sample size of N= 129 (Clark-Carter, 1997). Participants were sampled from the population of trainee clinical psychologists. This large accessible population enabled the use of a randomised control design and robust statistical analysis. The approximate population at the time of recruitment was N= 1,500 (estimated using information from the Clearing House for Postgraduate Courses in Clinical Psychology, 2011).

Twenty-five of the 30 course centres agreed to circulate the recruitment request and 309 trainees consented to participate. Trainees from the author's cohort were excluded. Ninety-two participants either did not start the survey or chose to exit before completion and were assumed to have withdrawn consent; 70.2% completed the survey (n= 217). Participants were aged 21-43; the majority (86%) were women reflecting the underlying population (in 2011, 81% accepted onto clinical training were women). See Table 1. for demographic information.

Materials

Vignette. Participants were presented with a description of 'client D' the information presented met the DSM-IV diagnostic criteria for major depression (American Psychiatric Association, 2000). The depression would most appropriately be described as "mild." Mild depression refers to depression which causes "mild functional impairment" and has "few, if any symptoms… in excess of the five required to make the diagnosis"" (NICE, 2009, p.62). The experimental manipulation was contained within the vignette and the symptoms were described as "typical of…" either "a biological

depression" or "a psychosocial depression." In the control condition no inferred aetiology was presented (see appendix D, p.110).

Survey tool. The online survey tool "SurveyMonkey®" was used to present the survey (see appendix E, p.112). Where appropriate, the presentation of the measures was randomised. The survey tool's prize draw function was utilised.

Questionnaires.

The modified survey of etiological beliefs and treatment effectiveness.

The survey consisted of two Likert scales: a 17-item, five-point, (1= definitely not a cause, 5= definitely a cause) measure of causal beliefs, and a 12-item, seven-point (1= definitely ineffective) to (7= definitely effective) measure of treatment effectiveness (Niewsu & Pepper 2010). The questionnaire was adapted from Goldstein & Roselli's (2003) original, by adding three items to the survey of etiological beliefs: "biochemical abnormalities," "recent misfortunes" and "disease in the brain." A principal component analysis on the data revealed a two components model; psychosocial factors (11-items, accounting for 20% of the variance) and biological (6-items, accounting for 19% of the variance). In the current study, scale reliability was acceptable to high (biological, α= 0.87; psychosocial, α= 0.66). Responses on the treatment effectiveness scale were divided into medical treatments (ECT, hospitalisation and anti-depressants, α= 0.57), psychological treatments (psychotherapy, cognitive therapy and behavioural therapy, α= 0.67), self-medication (alcohol and recreational drugs, α= 0.79) and self-initiated treatments (exercise, relaxation/yoga, self-help, and getting out more, α= 0.73).

Clinicians' attitude questionnaire - modified. An adapted version of the Clinicians Attitude Questionnaire (CAQ; Lam & Salkovskis, 2007) and General Attitude Questionnaire (Lam, Salkovskis & Warwick, 2005) was used. The modified questionnaire incorporated five items from the CAQ and four items from the GAQ.

Participants were asked to imagine they or a psychologist in their service was treating the client, and rate their responses to items such as "how likely do you feel the client would be to harm themselves?" on a 0-100% visual-analogue scale (transformed to a 1-11 scale for analysis) from "not at all" to "definitely." The original version of the CAQ had good test-retest reliability (r= 0.82).

A principal component analysis was conducted on the items used in this study (see appendix F, 121). Four components with eigenvalues greater than one were extracted, these accounted for 64.56% of the variance. The four factors were: "treatment success," combined from two items (curability and treatment effectiveness, loadings>.7), "engagement," combined from three items (motivation, drop-out and relapse, loadings>.57) "severity," combined from two items ("level of disability" and "intensity of treatment needed," loadings>.7), and "risk" combined from two items("likelihood to harm self" and "need for hospitalisation" (loadings>.47).

Perception of depression questionnaire (PDIQ), self-efficacy subscale. The 24-item self-efficacy subscale (PDIQ) was used as a measure of participants' beliefs about the client's control over the depression (Manber et al. 2003). The scale asks participant's to rate, along a four-point, Likert scale (1= Not at all, 4= very much so) how much the client would be able to use self-initiated strategies to control their depression (e.g. "making changes in their life"). The self-efficacy subscale has demonstrated good internal consistency (α= 0.91) and 8 week test-retest reliability (r= .83). Internal consistency in the current study was also high (α= .92).

Illness perception questionnaire modified for depression (IPQ), control-cure subscale. The six-item control-cure subscale (Fortune, Barrowclough & Lobban, 2004) was adapted from the Illness Perception Questionnaire (Weinman et al., 1996). The scale measures participant's beliefs about the ability to control or cure the depression,

along a five-point Likert scale, (1= strongly disagree; 5= strongly agree). Scale reliability was α= 0.56, and test-retest reliability was, r= .68 (Fortune et al, 2004). In the current study, reliability was also low (α= 0.54). The control subscale was higher (α= 0.62) and was used in the analysis.

Stigma Scale. The 25-item, five-point Likert scale, comprises five subscales (Nieuwsma & Pepper, 2010). The "authoritarianism" and "discrimination" scales were developed using Couture and Penn's (2003) review of the stigma literature. "Dangerousness" "dependency" and "affectivity" scales were created based on research by Angermeyer and Matschinger (2003). The stigma scale demonstrated high internal consistency (α= 0.9; subscales, α= 0.62-0.89, Nieuwsma & Pepper, 2010). The scale has convergent validity and correlates with the Internalised Stigma of Mental Illness Alienation Scale (Ritsher, Otilingam, & Grajales; r= 0.46, p<.01). In the current study the Stigma Scale and subscales demonstrated high internal consistency (α= .72-.93).

Procedure and ethics

Ethical approval was granted by the Salomons Ethics Panel at Canterbury Christ Church (appendix G, p.122). Two recruitment drives were carried out in February and June 2012, when all clinical psychology programmes on the Clearing House Website were contacted via email and/or phone. Recruitment emails (see appendix H-I, pp.123-124) were forwarded to clinical psychology trainees by course centres.

Participants were randomised to a condition and presented with the vignette. Participants then completed the study measures. Participants were able to register for a prize-draw (prizes of four vouchers worth up to £50). Registration details could not be linked to the data. The prize-draw was completed following data-collection and participants were notified of prizes via email. A full debrief and summary was emailed to participants following study completion (appendix J, p.126).

Results

Analyses

Analysis was conducted in two stages. Firstly, in-line with hypothesis one, the experimental data was analysed to see if the manipulation had an effect on causal beliefs. Potential moderator variables were then explored prior to testing the experimental hypotheses. The second section of the results presents the observational analysis. Results are presented in Tables 3 and 6. Main findings are discussed in the text.

Treatment of the Data

Normality was assessed by calculating skew and kurtosis values for all scales (see appendix K, p.123). Four scales were found to have significant levels of kurtosis and/or skew (values≥ +/-3; Kline, 2000). Examination of histograms indicated one extreme outliner in responses to the psychotherapy scale which was removed, correcting the distribution. A log transformation was conducted on the psychotherapy, risk and engagement scales correcting for kurtosis. Pearson's *r* was used to explore correlations between scales (all *r's*(217)<.50; see appendix L, p.130).

Where assumptions were met all data were analysed parametrically, using ANOVAs. Where hypotheses were made, *Post hoc* Tukey tests were used to make comparisons. *Post hoc* Bonferroni comparisons were used for single item analysis and for all exploratory analysis. Non-parametric data were analysed using Kruskal-Wallis (CI = 99%) and follow-up Mann-Whitney U tests.

Effect sizes are given using Pearson's *r* (small= .1, medium= .3, large= .5) and Cohen's *d* (small= 0.2, medium= 0.5, large= 0.8; Cohen, 1992). Effect sizes for ANOVA are given using ηp^2 (small= .01, medium= .06, large= .14, Kinnear & Gray, 2010).

Motivation Check

Motivation levels were tested by asking participants to identify symptoms which had occurred in the vignette (maximum correct= 8). There appeared to be high motivation across the sample (M= 6.77, SD= 1.25).

Experimental Groups

Differences across groups were analysed using X^2 tests for categorical variables and ANOVA for continuous variables. Withdrawal from the study did not vary across conditions, $X^2(2, N= 92) = .93$, $p = .65$. The number of depression symptoms identified from the vignette varied across conditions, $F(2,217) = 3.99$, $\eta p^2 < .02$, $p < .02$. Participants in the control condition identified more symptoms ($M = 7.15$, $SD = 1.25$) than either the biological group ($M = 6.62$, $SD = 1.26$, $p < .05$) or the psychosocial group ($M = 6.62$, $SD = 1.25$, $p < .05$). This was a small effect suggesting differential motivation across groups was not a concern.

There were no significant differences across conditions in terms of demographics, year of training or the number of people who identified as having a theoretical orientation. Nor were there any significant differences in participants' underlying causal beliefs about mental illness (see Table 1 and 2).

Table 1.

Demographic details and group differences (X^2)

		All	Biological	Psychosocial	Neutral	X^2	*df*
		n	n	n	n		
Sample		217 (100)	85 (39)	71 (33)	61 (28)		
Gender						0.87	2
	Male	31 (14)	14 (16)	8 (11)	9 (15)		
	Female	186 (86)	71 (84)	63 (89)	52 (85)		
Year of training						3.48	4
	1	66 (30)	21 (25)	26 (37)	19 (31)		
	2	80 (37)	34 (40)	26 (37)	20 (33)		
	3	71 (33)	30 (35)	19 (27)	22 (36)		
Ethnicity						21.30	22
	White British	164 (76)	63 (74)	53 (75)	48 (79)		
	White other	30 (14)	9 (11)	13 (18)	8 (13)		
	Black British	2 (1)	1 (1)	0 (0)	1 (2)		
	Black other	4 (2)	1 (1)	1 (1)	2 (3)		
	Asian	4 (2)	1 (1)	1 (1)	2 (3)		
	Mixed race	7 (3)	4 (1)	2 (3)	1 (2)		
	Not stated	6 (3)	4 (5)	1 (1)	1 (2)		
Identified theoretical orientation						6.60	4
	Yes	53 (24)	24 (28)	15 (21)	14 (23)		

Note. Percentages are given in parentheses. Cumulative percentages may not add up to 100% due to rounding.

Table 2.

Group differences (ANOVA)

Instrument		All (Mean)	Biological (Mean)	Psychosocial (Mean)	Neutral (Mean)	*F*	*df*
	Age	28.48 (3.60)	28.45 (3.60)	28.45 (4.00)	28.57 (3.13)	0.26	2,214
Causal beliefs about mental illness - SEB							
	Biological	2.54 (1.01)	2.55 (0.96)	2.58 (1.00)	2.49 (1.11)	1.20	2,214
	Psychological	3.6 (0.89)	3.65 (0.89)	3.52 (0.89)	3.75 (0.87)	1.22	2,214
	Environmental	3.74 (0.86)	3.74 (0.94)	3.65 (0.85)	3.84 (0.76)	0.78	2,214

Note. SEB= Survey of Etiological Beliefs, M= Mean, Standard deviations are presented in parentheses below means.

Outcome of the Experimental Manipulation

There was a non-significant trend in the predicted direction, with a small effect of group on biological causal ratings (p<.09). The between group differences did not reach significance, although participants in the biological condition gave higher ratings of biological causality than participants in the psychosocial group (d= 0.28, p= .19) or control group (d= 0.33, p= .13). There was no difference between the psychosocial and control group (d<0.05, p= .97).

Due to the divergence of causal factors making up the biological (e.g. chemical imbalance vs. brain disease) and psychosocial scales (e.g. negative life events vs. will power) analysis was conducted on individual items. There was a small effect of group on ratings of "biological factors" (p<.01). Participants in the biological condition rated biological factors as more likely to be causal in the clients' depression than participants in the psychosocial condition (d= 0.49, p<.01).

There was a small effect of group on ratings of "general stress," (p= .02) and "response to a negative life event," (p= .03). Participants in the biological condition were less likely to rate the depression as being caused by general stress (d= 0.42, p= .02) compared to people in the psychosocial condition. There was a trend towards people in the biological group viewing a negative life event as being less likely to be causal in the depression, relative to the psychosocial group (d= 0.34, p= .078) or the neutral group (d= 0.38, p= .08).

There were no significant between-groups effects on any other scale items, F's(2,214)= 0.5-2.1, p>.20, ηp^2<.02, P<0.45.

Potential moderators. ANOVA's were conducted to test for moderators of the effect of group x causal beliefs. Potential moderators were proposed to be: participant's underlying beliefs about mental illness, gender, year of training and whether or not participants identified as having a theoretical orientation. There were no significant interactions (see appendix M for a description of this analysis, p.130).

Summary. The findings suggest that the experimental manipulation had a small effect on causal beliefs about the client's depression. Framing the depression as "typical of a biological depression" slightly increased ratings of biological causality; however the difference between conditions did not reach significance. Analysis of individual items showed significant variability in causal ratings across groups. In line with the hypothesis, reading the biological vignette increased participant's causal ratings of biological factors, and reduced causal ratings of stress and negative life events. The effect was not moderated by participants' gender, year of training, identification with a theoretical orientation or their underlying causal beliefs about mental illness.

This bias in causal attributions only occurred in the biological condition; there was no effect of framing the client's symptoms as "typical of a psychosocial depression."

Figure 1. highlights the similarity in causal ratings across the psychosocial and control group. In contrast, there were large differences between the biological and psychosocial groups. For example, 47% of the biological group agreed that biological factors were likely to be a leading cause of the client's depression, compared to 23% of the psychosocial group. In turn, 41% of the biological group felt negative life events were a leading cause of the depression compared to 61% of the psychosocial group. A notable exception was for ratings of psychological factors.

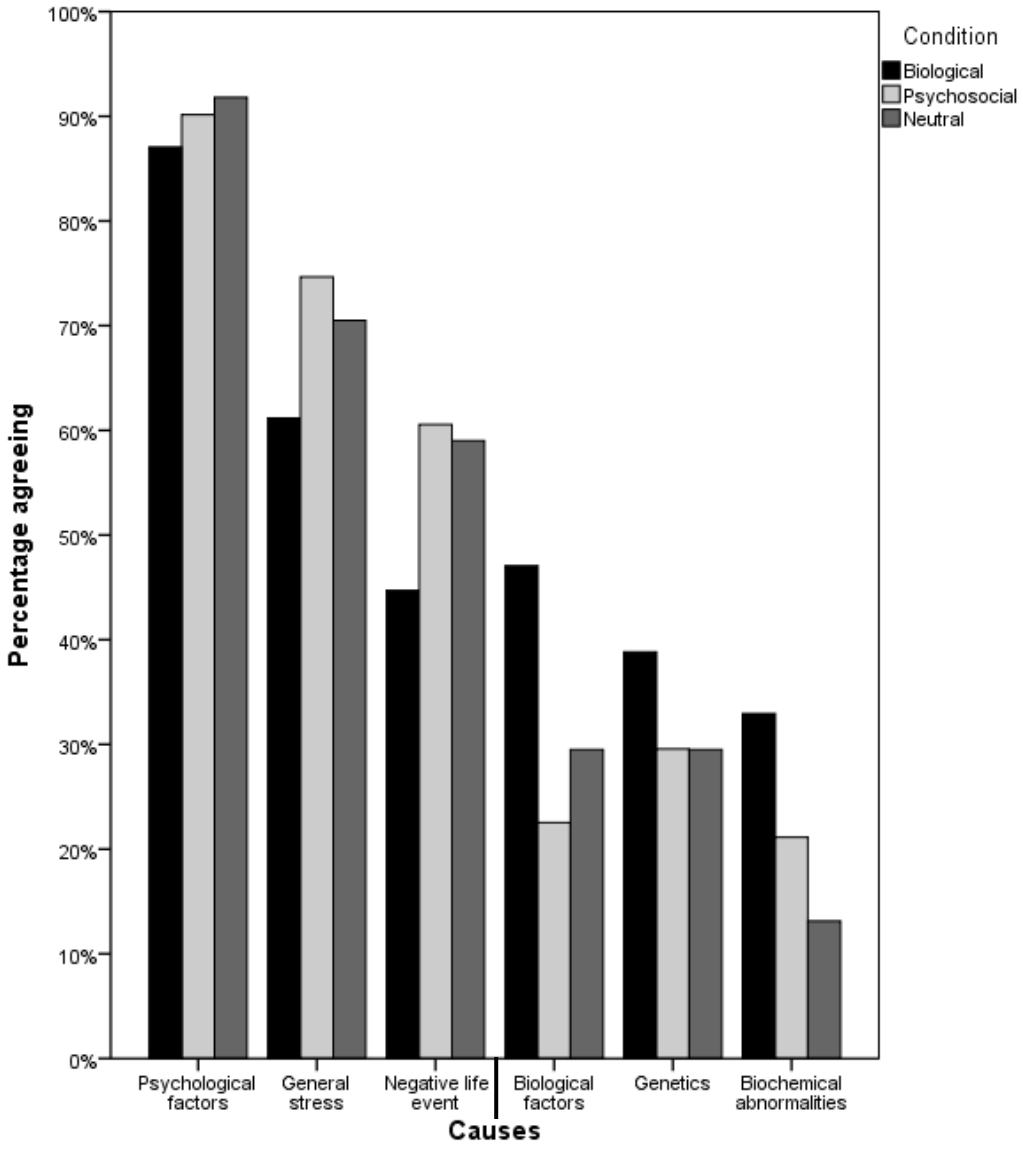

Figure 1.

Graph showing the percentage of people, across groups, who believed each cause was leading in the client's depression.
Note. Only the three most popular biological and psychosocial causes are represented

Experimental Hypotheses

Treatment effectiveness. Hypothesis 2 was partially supported. There was a small effect of group on perceived effectiveness of medical treatments (p= .03). Participants in the biological group were more likely to endorse medical treatments than the neutral group (d= 0.43, p= .03). Participants in the biological group also rated medical treatments as more effective than participants in the psychosocial group. However, this difference did not reach significance (d= 0.30, p= .13.).

The effect of group on "anti-depressants," "ECT" and "hospitalisation" were analysed separately. There was no effect of group on perceived effectiveness of anti-depressants, F(2,214)= 0.76, p= .49, P= .18. Agreement that anti-depressants would be effective was high across conditions (≥92%).

There was a marginal effect of group on hospitalisation, F(2,214)= 2.95, p= .055, ηp^2= .03, P= 0.60, with people in the biological group rating hospitalisation as more effective than those in the neutral group (d= 0.37, p= .053). There was no significant difference between the biological and psychosocial group (d= 0.22, p= .47).

There was a non-significant trend towards an effect of group on ratings of effectiveness for ECT, $h(2)$= 4.85, p= .09, CI= .08-1.00. Mann-Whitney follow-up tests indicated this trend was in the predicted direction. Trainees in the biological condition rated ECT as more effective (mdn= 2, moderately ineffective) in treating the client's depression than those in the psychosocial group (mdn= 1, definitely ineffective), U= 2572, z= -1.75, p= .08, r= -.14, or the neutral group (mdn= 1), U= 2155, z= -1.92, p<.06, r= -.16. There was no difference between the psychosocial and neutral group (U= 2572,

z= -.24, *p*= .81). Survey responses indicated that 37% of the biological group rated ECT as possibly effective, compared to 27% of the psychosocial group and 25% of the control group.

In contrast to the hypothesis, there was no significant effect of group on perceived effectiveness of "psychological therapy," $F(2,213)= 0.48$, $p= .62$, P= 0.13. Average ratings for psychological therapy were in the moderately effective range, with 100% of participants agreeing that psychological therapy would be (at least) possibly effective in treating the client's depression (see Figure 2).

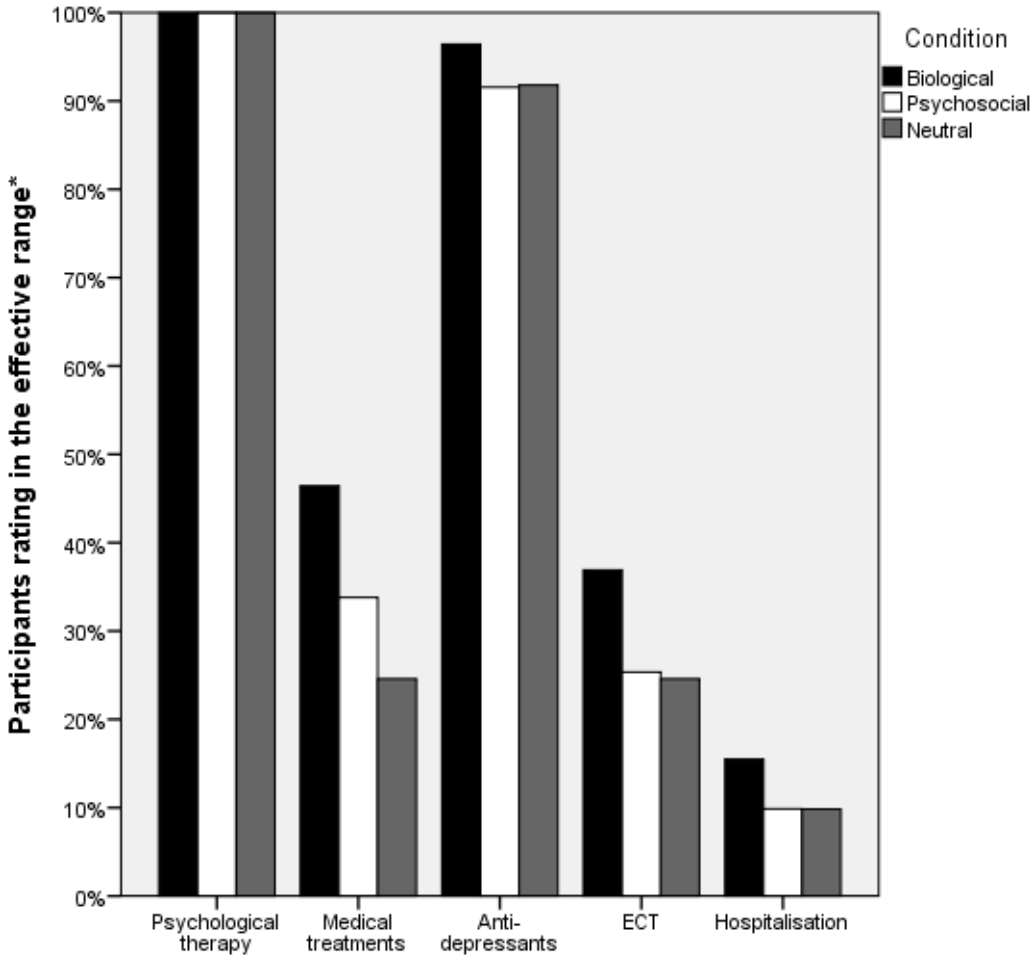

Figure 2.

Graph showing the percentage of people, across groups, who perceived each treatment to be effective in treating the client's depression.

* "Not sure/possibly" ratings were collapsed into the effective category

Table 3.

Between-groups effects (ANOVA results)

Instrument (Range)		**Biological** $\bar{x}$	**Psychosocial** $\bar{x}$	**Neutral** $\bar{x}$	**F**	**df**	**ηp^2**	**β**
SEB (1-5)	Overall biological	2.89 (0.69)	2.70 (0.66)	2.67 (0.64)	2.40 ǂ	2,214	.02	0.48
	Overall psychosocial	3.18 (0.42)	3.26 (0.35)	3.31 (0.34)	2.47	2,214	.02	0.44
	"Biological factors"	3.32[a] (0.82)	2.93[b] (0.76)	3.07[ab] (0.80)	4.97**	2,214	.04	0.80
	"Stress"	3.60 [a] (0.68)	3.89[b] (0.69)	3.79[ab] (0.67)	3.80*	2,214	.03	0.64
	"Negative life event"	3.32 (0.89)	3.59 (0.69)	3.61 (0.59)	3.47*	2,214	.03	0.08
STE (1-7)	Medical	3.16[a] (0.89)	2.89[b] (0.91)	2.80[b] (0.80)	3.61*	2,214	.03	0.66
	Psychological therapy	6.01 (0.67)	6.01 (0.71)	5.90 (0.71)	0.48	2,213	<.01	0.13
	Self-medication	1.49 (0.95)	1.46 (0.80)	1.52 (0.89)	0.93	2,214	<.01	0.60
	Self-initiated	5.49 (0.73)	5.42 (0.55)	5.47 (0.62)	0.91	2,214	<.01	0.77
PDQ (1-4)	Self-efficacy	2.73 (0.44)	2.71 (0.41)	2.69 (0.44)	0.43	2,214	<.01	0.07
IPQ-D (1-6)	Control-cure	4.23 (0.56)	4.21 (0.45)	4.21 (0.43)	0.35	2,214	<.01	0.06
	Control	4.29 (0.49)	4.25 (0.55)	4.28 (0.53)	0.11	2,214	<.01	0.07
Stigma (1-5)	Global	3.19 (0.62)	3.23 (0.41)	3.10 (0.64)	0.91	2,214	<.01	0.21
	Authoritarian	3.30 (0.67)	3.43 (0.47)	3.18 (0.70)	2.85	2,214	.03	0.55
	Discrimination	3.29 (0.83)	3.32 (0.69)	3.17 (0.86)	0.57	2,214	<.01	0.14
	Dangerousness	2.39 (0.65)	2.34 (0.58)	2.31 (0.66)	0.27	2,214	<.01	0.09
	Affectivity	3.52 (0.71)	3.59 (0.47)	3.48 (0.71)	0.44	2,214	<.01	0.12
	Dependency	3.53 (0.90)	3.33 (0.81)	3.25 (0.91)	1.97	2,214	.02	0.40
CAQ-M (1-11)	Treatment effectiveness	8.23 (1.26)	8.35 (1.25)	8.21 (1.10)	2.63	2,214	<.01	0.09
	Engagement	6.91 (1.27)	7.05 (1.25)	6.94 (1.13)	0.55	2,214	<.01	0.14
	Perceived risk	2.70 (1.38)	2.68 (1.19)	2.99 (1.26)	1.49	2,214	.02	0.32
	Severity	3.21 (1.25)	3.98 (1.23)	3.13 (1.20)	2.07	2,214	.01	0.42

Note. Standard deviations are given in parentheses under means. SEB= Survey of Etiological Beliefs; STE= Survey of Treatment Effectiveness; PDQ= Perceptions of Depression Questionnaire; IPQ-D= Illness Perceptions Questionnaire–Depression, CAQ-M= Clinicians Attitude Questionnaire-Modified.
* $p \leq .05$,** $p \leq .01$, # $p \leq 0.8$, means with differing subscripts are significantly different at $p \leq .05$.
.

Control. There was no effect of group on measures of control or self-efficacy (*p's*≥.90). Thus the hypothesis, that people in the biological group would see the depression as less controllable was not supported. The majority of participants 98-100% "agreed" the depression could be controlled and perceived the client to be "somewhat" able to demonstrate self-efficacy in managing the depression.

Clinicians' attitudes. The hypothesis that trainees in the biological condition would hold more pessimistic attitudes than participants in the psychosocial condition was not supported. There were no significant effects of group on ratings of "treatment effectiveness," "engagement," "severity" or "risk" (all *p's*>.13). Across conditions risk and severity were rated low, engagement was rated moderately and the likelihood of treatment being effective was perceived to be high.

Stigma. Overall, the hypothesis that condition would affect judgements of perceived stigma was not supported. There was no effect of group on measures of "overall stigma," "dangerousness," "dependency," or "discrimination" (all *p's*≥.14). There was a marginal effect of group on "authoritarian" stigma *(p*= .058). Participants in the psychosocial group were more likely to perceive others as holding authoritarian attitudes to the client than participants in the neutral group (*d*= 0.42, *p*<.05). There was no significant difference between the biological and psychosocial group (*d*= 0.23, *p*>.10).

The majority of participants, across conditions, felt the client was at risk of experiencing most types of stigma (60%-90%). The only exception to this was stigma related to perceptions of dangerousness (8%). The data suggests that participants in

the psychosocial condition were more likely to feel the client would experience stigma, compared to the biological condition (9% difference). A notable exception was "dependency," with 10% more people in the biological condition believing the client would be seen as needy and dependent. The lowest stigma ratings, across all scales, were given by participants in the neutral condition (see Figure 9, appendix N, p.132),

Observational Findings

Causal beliefs about the depression. Participants were divided into one of four groups based on their ratings on the SEB: those who scored higher than the mean on the biological scale (≥2.84; "biological"), those who scored higher than the mean on the psychosocial scale (≥3.20; "psychosocial"), those who scored higher than the mean on both scales ("biopsychosocial") and those who scored lower than the mean on both scales ("low causal"). The resulting groups reflected participants' primary causal beliefs about the clients' depression.

Group differences. There were no significant differences across groups in terms of the experimental group they had been allocated to (p= .47) or in the test of motivation, F(3,213)= 0.31, np^2= .01, p= .82, P= 0.11. Nor were there any significant differences across groups in gender, year of training or ethnicity ($p's$≥.11). There was a significant difference across groups in the number of people who identified with a theoretical orientation (p<.02). Group frequencies indicated that participants with strong biological beliefs were less likely to identify with a theoretical orientation (13% vs. 25-33%). There was a significant effect of group on age, with people with "low causal" beliefs being on average two years older than people in the psychosocial group (std. error= .67, p<.02).

There was a significant effect of group on endorsement of psychological, environmental and biological factors as important causes of mental illness (all p's≤.05).

Post hoc tests indicated that causal beliefs about mental illness were congruent with participants' causal attributions about the client's depression (See table 5).

Table 4

Group differences: Aetiological beliefs about the client's depression (X^2)

		All	Biological	Psychosocial	Biopsychosocial	Low causal	X^2	df
		n	n	n	n	n		
Sample		217 (100)	56 (26)	59 (27)	49 (23)	53 (24)		
Condition							5.57	6
	Biological	85 (39)	26 (31)	19 (22)	19 (22)	21 (25)		
	Psychosocial	71 (33)	20 (28)	21 (30)	13 (18)	17 (24)		
	Control	61 (28)	10 (16)	19 (31)	17 (19)	15 (25)		
Gender							1.41	3
	Male	31 (14)	6 (11)	10 (17)	6 (12)	9 (17)		
	Female	186 (86)	50 (89)	49 (83)	43 (88)	44 (83)		
Year of training							10.43	6
	1	66 (30)	23 (41)	14 (24)	17 (35)	12 (23)		
	2	80 (37)	20 (36)	23 (29)	20 (41)	17 (32)		
	3	72 (33)	13 (23)	22 (37)	12 (25)	24 (45)		
Ethnic origin							26.50	33
	White British	164 (76)	40 (71)	47 (80)	35 (71)	42 (79)		
	White other	30 (14)	11 (20)	4 (7)	9 (18)	6 (11)		
	Black British	2 (1)	0 (0)	1 (2)	1 (2)	0 (0)		
	Black other	(2)	0 (0)	2 (3)	1 (2)	1 (2)		
	Asian	4 (2)	1 (2)	1 (2)	1 (2)	1 (2)		
	Mixed race	7 (3)	2 (4)	3 (5)	1 (2)	1 (2)		
	Not stated	6 (3)	2 (4)	1 (2)	1 (2)	2 (4)		
Identify with a theoretical orientation							15.42*	6
	Yes	53 (24)	7 (13)	17 (29)	16 (33)	13 (25)		

Note. Cumulative Percentages may not add up to 100% due to rounding, percentages are given in parentheses.
*$p \leq .05$

Table 5.

Group differences: Aetiological beliefs about the client's depression (ANOVA)

Instrument (Range)	**Item**	**All**	**Biological**	**Psychosocial**	**Biopsychosocial**	**Low causal**	**F**
		Mean	Mean	Mean	Mean	Mean	
	Age		27.81 (2.89)	28.17 (3.50)	28.14 (3.18)	29.90 (4.41)	3.71**
SEB - beliefs about mental illness							
	Biological	2.54 (1.01)	2.89^{ac} (0.91)	2.12^{bcd} (1.04)	2.82^{abc} (1.07)	2.40^{bd} (0.84)	7.93**
	Psychological	3.65 (0.89)	3.55^{ab} (0.87)	3.90^{a} (0.80)	3.67^{ab} (0.97)	3.45^{b} (0.87)	2.72*
	Environmental	3.74 (0.86)	3.57^{a} (0.83)	4.00^{b} (0.79)	3.65^{ab} (0.93)	3.70^{ab} (0.87)	2.77*

Note. SEB= Survey of Etiological Beliefs, standard deviations are presented in parentheses below means, means with differing subscripts are significantly different at $p \leq .05$.
*$p \leq .05$, ** $p \leq .01$

Main Effects of Causal Beliefs

Treatment effectiveness. There was a large effect of causal beliefs on perceived effectiveness of "medical treatments," ($p < .001$). Participants in the biological group were more likely to endorse medical treatments than the psychosocial group ($d = 1.12$, $p > .001$) and the "low causal" group ($d = 0.63$, $p < .001$). Participants in the biopsychosocial group also rated medical treatments to be more effective than the psychosocial group ($d = 0.99$, $p < .0001$) and the "low causal" group ($d = 0.54$, $p = .02$).

There was a large effect of group on ratings of effectiveness for "anti-depressants" ($p < .001$). Participants in the psychosocial group perceived anti-depressants to be less effective than participants in the biological group ($d = 1.13$,

p<.001), biopsychosocial group (*d*= 0.68 *p*= .001) or "low causal" group (*d*= 0.46, *p*= .058).

There was a medium effect of group on "hospitalisation," (*p*<.01). Those in the psychosocial group perceived hospitalisation to be less effective in treating the clients depression than the biological group (*d*= 0.67, *p*<.02) and the biopsychosocial group (*d*= 0.58, *p*= .02).

There was a medium effect of group on ECT, *h(3)*= 15.5, *p*<.001, *CI*<0.00-0.01. The ratings of effectiveness for ECT were significantly higher in the biological group (mdn= 2; moderately ineffective) compared to the psychosocial group (mdn= 1; definitely ineffective), *U*= 1120; *z*= -3.35, *p*<0.01, *r*= -.31, and the "low causal" group (mdn= 1), *U*= 1112; *z*= -2.52, *p*= 0.01, *r*= -.24.

Ratings of effectiveness for ECT in the biopsychosocial group were also significantly higher than the psychosocial group (mdn= 2), *U*= 946.5; *z*= -3.46, *p*<.01, *r*= -.33, and the "low causal" group U= 936.5; *z*= -2.70, *p*<.01, *r*= -.27.

There were no significant differences between the biological and biopsychosocial group *U*= 1292; *z*= -.552, *p*= .58, or between the "low causal" group and the psychosocial group *U*= 1496.5; *z*= -.486, *p*= .63, on effectiveness ratings for ECT.

Overall, 87% of the psychosocial group and 76% of the low causal group rated ECT as ineffective compared to 57% of the biological group and 53% of the biopsychosocial group.

Table 6.

Between-group effects: Causal beliefs about the client's depression

Instrument (Range)		**Biological** $\bar{x}$	**Psychosocial** $\bar{x}$	**Biopsychosocial** $\bar{x}$	**Low causal** $\bar{x}$	**Statistic** ***F***	***df***	***ηp²***	***β***
STE (1-7)									
	Medical	3.34[a] (0.84)	2.50[b] (0.64)	3.29[a] (0.93)	2.81[b] (0.84)	13.61**	3,213	.16	1.00
	Anti-depressants	5.59[a] (0.73)	4.51[b] (1.14)	5.27[a] (1.08)	5.00[a] (1.00)	12.00**	3,213	.15	1.00
	Hospitalisation	2.00[a] (0.89)	1.42[b] (0.83)	2.00[a] (1.14)	1.60[b] (0.97)	4.45**	3,213	.06	0.87
	Psychological therapy	6.00 (0.69)	6.04 (0.61)	6.06 (0.74)	5.83 (0.73)	1.23	3,212	.02	0.13
	Self-medication	1.32 (0.61)	1.33 (0.61)	1.77 (1.18)	1.58 (0.99)	2.67*	3,213	.04	0.65
	Self-initiated	5.56 (0.55)	5.51 (0.69)	5.48 (0.81)	5.27 (0.67)	1.84	3,213	.03	0.47
PDQ (1-4)	Self-efficacy	2.70[a] (0.39)	2.78[a] (0.41)	2.85[a] (0.45)	2.55[b] (0.42)	4.61**	3,213	.06	0.89
IPQ-D (1-5)	Control	4.33 (0.48)	4.33 (0.44)	4.36 (0.56)	4.09 (0.67)	2.92*	3,213	.04	0.69
Stigma (1-5)	Global	3.17 (0.54)	3.24 (0.52)	3.25 (0.51)	3.05 (0.69)	1.46	3,213	.02	0.38
	Authoritarian	3.32 (0.68)	3.38 (0.54)	3.38 (0.52)	3.17 (0.72)	1.46	3,213	.02	0.38
	Discrimination	3.23 (0.75)	3.35 (0.73)	3.40 (0.73)	3.09 (0.94)	1.55	3,213	.02	0.14
	Dangerousness	2.38 (0.55)	2.40 (0.69)	2.40 (0.66)	2.21 (0.61)	1.09	3,213	.02	0.29
	Affective	3.53 (0.62)	3.55 (0.56)	3.61 (0.56)	3.43 (0.79)	0.68	3,213	.01	0.19
	Dependency	3.25 (0.86)	3.56 (0.75)	3.40 (0.93)	3.33 (0.98)	1.29	3,213	.02	0.34
CAQ-M (1-11)	Treatment effectiveness	8.12 (1.31)	8.36 (1.29)	8.41 (1.10)	8.18 (1.10)	1.38	3,213	.01	0.69
	Engagement	6.67[a] (1.18)	7.34[b] (1.28)	7.06[ab] (1.09)	6.78[ab] (1.21)	3.62**	3,213	.05	0.79
	Perceived risk	2.94 (1.25)	2.49 (1.12)	3.07 (1.71)	2.63 (0.97)	3.08	3,213	.03	0.72

Severity	4.49 (1.06)	4.58 (1.15)	4.67 (0.78)	4.30 (0.95)	1.60	3,213	.02	0.42

Note. Standard deviations are given in parentheses under means, STE= Survey of Treatment Effectiveness; PDQ = Perceptions of Depression Questionnaire; IPQ-D= Illness Perceptions Questionnaire – Depression; CAQ-M = Clinicians Attitude Questionnaire-Modified, means with differing subscripts are significantly different at p≤.05.
* $p \leq .05$, ** $p \leq .01$,

There were no significant effects of group on "self-initiated interventions" (p= .14). Nor were there any between-group effect on ratings of "psychological therapy," (p= .30). As can be seen from Figure 3, there was a ceiling effect for psychological therapy with all participants, across groups, rating it in the effective range.

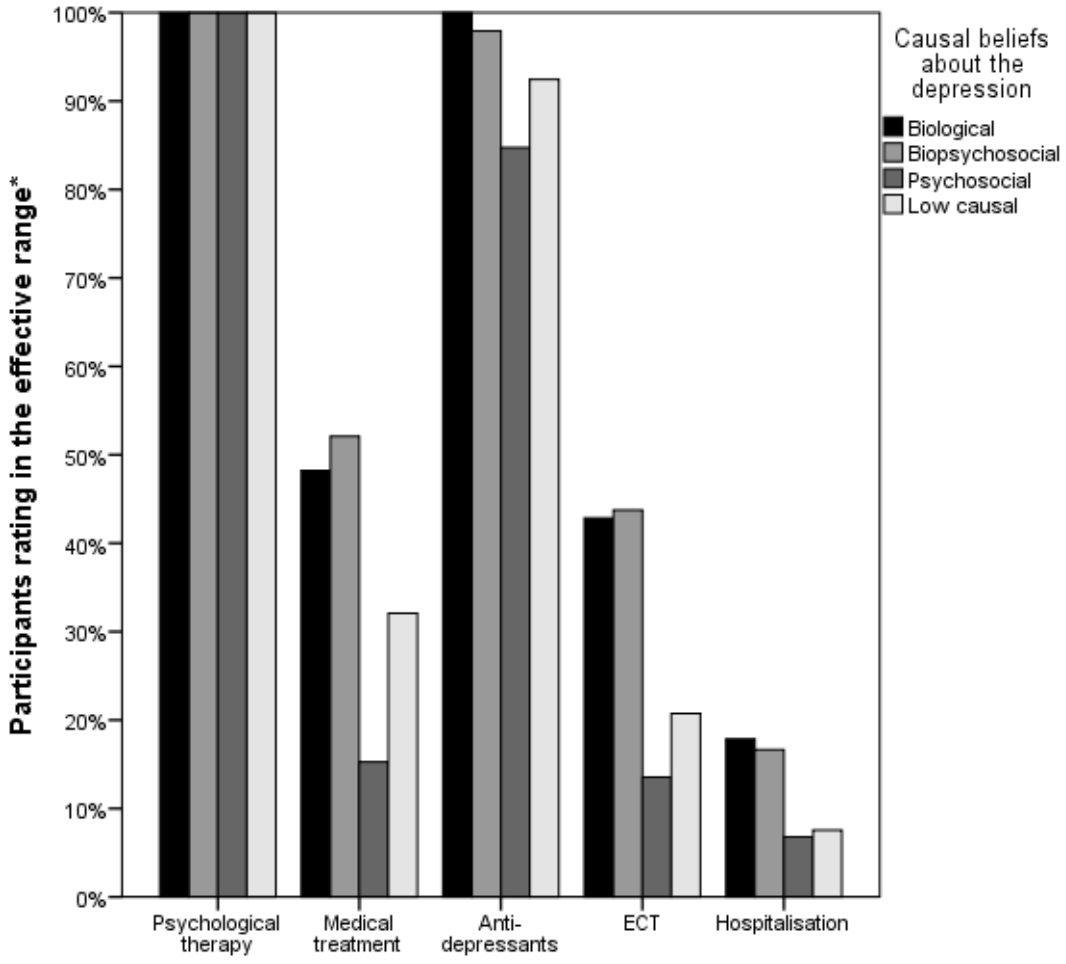

Figure 3.

Percentage of participant's whose ratings for each treatment fell in the effective range.

Note

* “Not sure/possibly” ratings were included in the effective range.

Control. There was a medium effect of group on control (p<.01). There was a non-significant trend towards participants in the low causal group rating the depression as less controllable compared to the biopsychosocial group, d= .47, p= .07. Participants in the low causal group also rated the depression as less controllable than participants in the psychosocial group (d= 0.42) and the biological group (d= 0.41), although these differences did not reach significance (p>.11).

A small effect of group on self-efficacy was also found (p<.05). Participants with “low causal” beliefs rated the client as having less self-efficacy than participants in the biopsychosocial group (d= .69, p= .002), and marginally less than the psychosocial group (d= 0.55, p= .062). There was no significant difference between the biological group and the low causal group (d= 0.37, p= .34).

These findings suggest that holding a causal model of the client’s depression is important in perceiving that the depression can be controlled. In addition, strongly endorsing psychosocial causes’ either alone or as part of a “biopsychosocial model” increases perceptions that the client is able to implement strategies to manage their depression (see Figure 4).

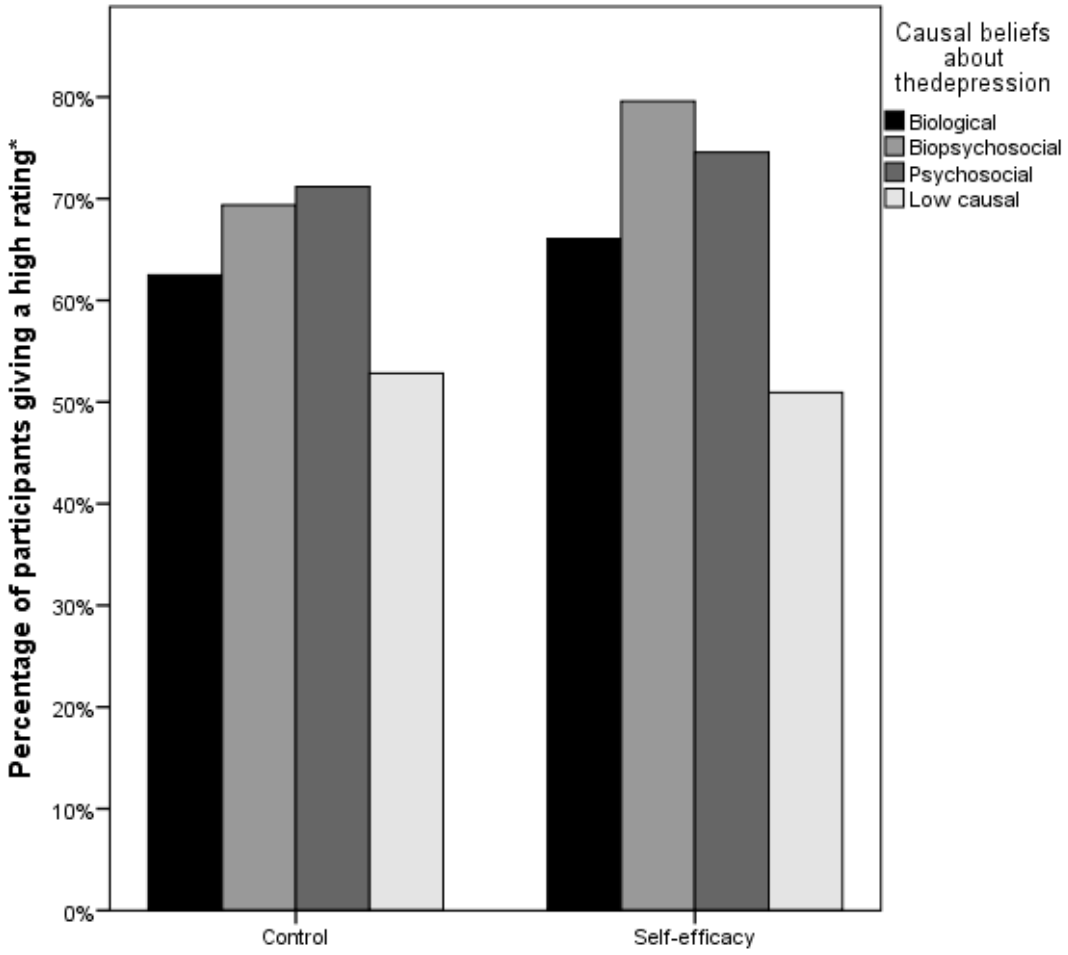

Figure 4.

Percentage of participants rating the depression as highly controllable (*ratings >4) and the client as highly able to demonstrate self-efficacy over the depression (*ratings>2.5).

Clinicians' attitudes. There was a medium effect of causal beliefs on ratings of client "engagement," (p= .014). The psychosocial group rated engagement as significantly higher than the biological group (d= 0.54, p= .018). There was a trend towards participants in the psychosocial group having higher ratings of engagement than the low causal group (d= 0.45, p= .086). Fifty-six per cent of participants with high psychosocial beliefs rated client engagement in therapy as high, compared to 45% of

people with strong biopsychosocial beliefs, 36% of people with strong biological beliefs and 36% of people with low causal beliefs (see figure 5).

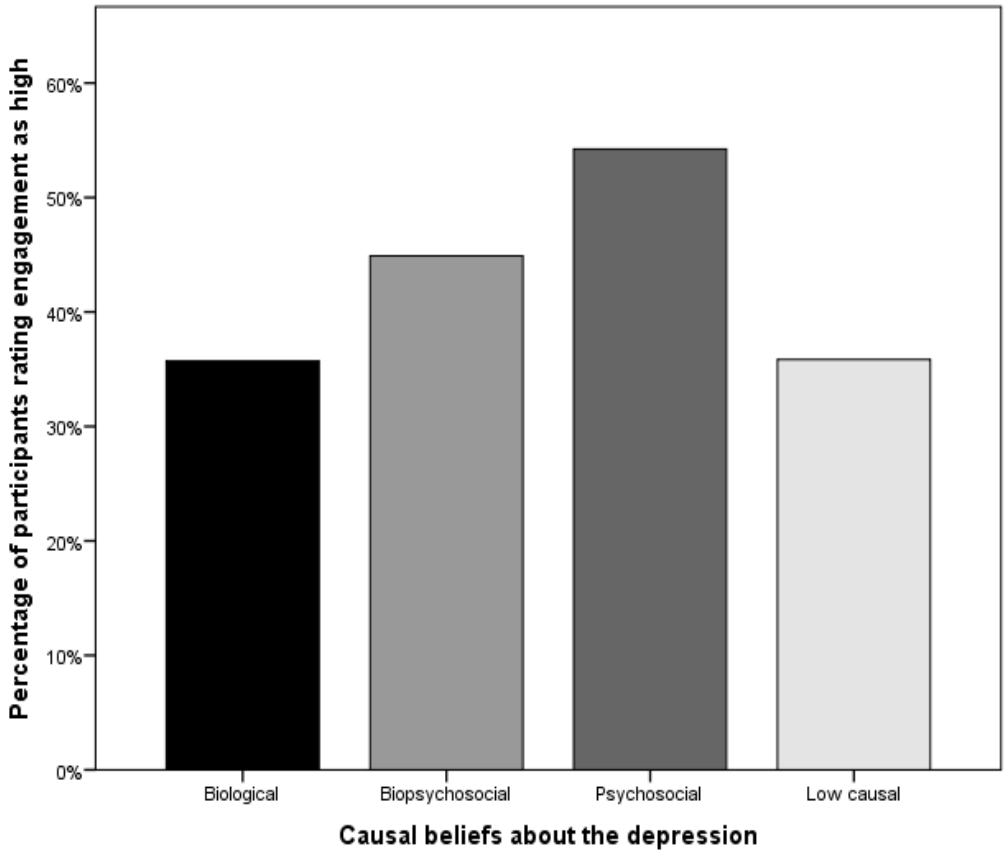

Figure 5.

Percentage of participant's rating the client's motivation as high (>60%).

There was a marginal effect of group on "risk" (p= .058). Participants with strong psychosocial beliefs perceived the client to be less risky than those in the biopsychosocial group (d= 0.40, p= .14) or the biological group (d= 0.38, p= .14). These were small effects which did not reach significance. Overall, risk was rated as low (<20%). See figure 6 for a break-down of risk ratings as function of causal beliefs.

There was no significant effect of group on "treatment effectiveness," or "severity" (*p's*≥.19*).* Perceptions of treatment effectiveness were high across the sample (ratings>70%) and severity of the depression was moderately low (ratings<40%).

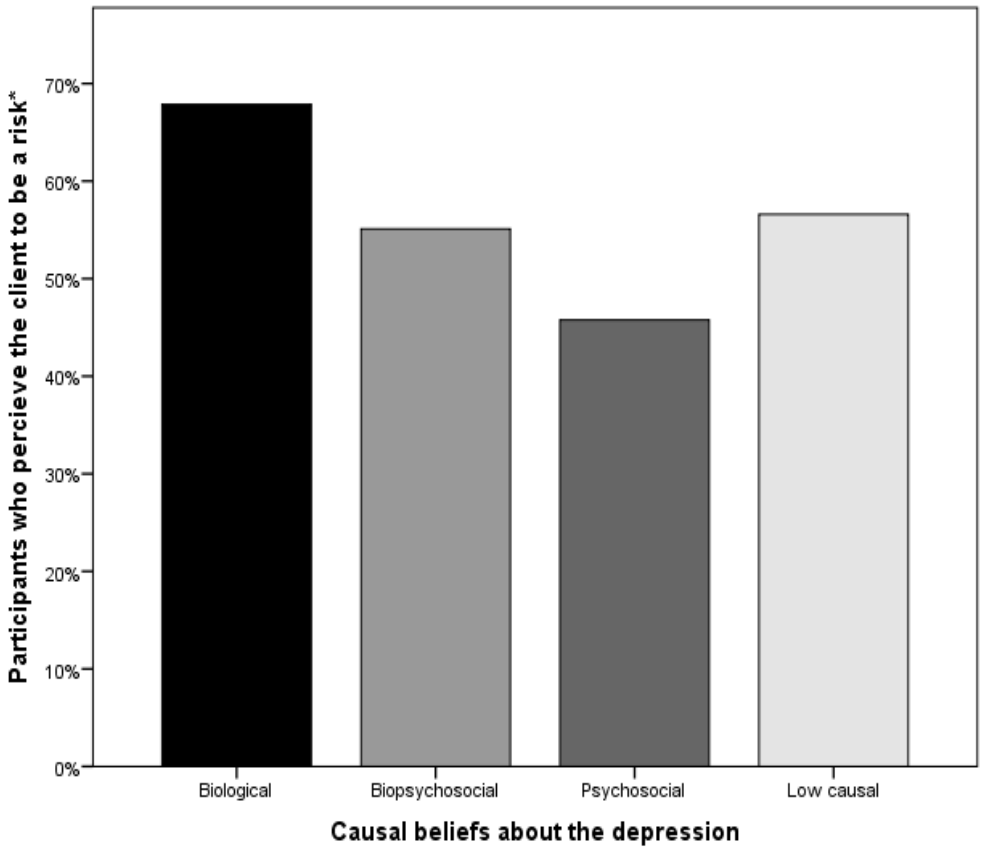

Figure 6.

The percentage of participants across groups who rated the client as being a risk (ratings≥10%).

Stigma. There was no effect of participants causal beliefs about the client's depression on measures of "overall stigma," "dangerousness," "discrimination," "dependency," "affective stigma" or "authoritarian" stigma (all *p's*≥.20).

Discussion

In line with previous experimental findings (Ahn et al., 2009), the data suggests that trainee clinical psychologist's causal beliefs were biased by presenting the depression as biological. This effect was small and only evident on the most endorsed causes. The effect is of note, given the very subtle nature of the manipulation. The client's depression was not stated to be caused by biological factors, rather it was framed as "typical of biological depression," and the vignettes described identical symptoms. Despite this, trainees who read that the depression was "typical of a biological depression" felt the depression was more likely to be caused by biological factors and less likely to be caused by stress or negative life events. Although this assertion may seem logical, there is an absence of evidence for a biological-psychosocial distinction in depression (Pies, 2009; Hasler, 2010).

Presenting the depression as biological was also found to increase trainees' perceptions of the effectiveness of medical treatments. This is consistent with previous findings (Kuyken et al. 1992; Ahn et al., 2009). The effect of the current study was small. Further research is warranted to examine whether the effect of framing depression as biological is amplified in real-life settings. This is of clinical significance, as referrals, and clients themselves, may state that the depression is biological, especially when recurrent or if there is a family history. If clinicians can be biased by this information it may lead them to advocate for medical treatments when this is not indicated by client information or NICE guidance (2009). In contrast to Ahn et al. (2009), this study did not find an effect of presenting the depression as biological on the perceived effectiveness of psychological therapy. Psychological interventions were rated as highly effective across all conditions suggesting there may have been a ceiling effect, which would be unsurprising given the professional training of the sample used.

There was no effect of framing the depression as psychosocial on causal beliefs or treatment effectiveness and the reason for this is unclear. Psychosocial causal factors were more strongly endorsed than biological factors across the sample, thus the manipulation may have had less room to have an effect. It is also possible that "biological depression" was felt to be a more credible subtype of depression compared to psychosocial depression and therefore had more influence on causal beliefs.

In contrast to previous findings in lay populations, this study found no effect of framing the depression as biological on perceptions of self-efficacy or control (Goldstein & Roselli, 2003; Brown et al., 2007, Deacon & Baird, 2009). Clinicians may be less likely to see biological causes of depression as untreatable or uncontrollable, compared to non-professionals.

Perceptions of stigma towards the client were rated to be high, across conditions, suggesting that stigma in depression is a significant concern. Labelling the depression as psychosocial led clinicians to feel the client would be more likely to experience authoritarian stigma, such as others perceiving them to be incompetent. Agreement that the client would experience stigma (overall), was also higher in the psychosocial condition. Previous findings have been mixed, some studies have found that psychosocial explanations for depression increase stigma (Goldstein & Roselli, 2003) whereas other studies have shown no effect (Nieuwsma & Pepper, 2010) or the reverse (Kent & Read, 1998). Perceptions of stigma were the lowest in the neutral group suggesting that additional labelling of the depression as biological or psychosocial increased stigmatising attitudes. This is in contrast to previous research which suggests causal explanations reduce judgements of stigma (Rusch et al., 2009; Kim & LoSavio, 2000). Further research is needed to see if these are significant and reliable effects.

Exploratory analysis of the data found that trainees were heavily biased by their own causal models of the depression. There were large effects of endorsing primarily biological (or biopsychosocial) models on perceived effectiveness of medical treatments. Participants who had strong biological causal beliefs felt anti-depressants, ECT, and hospitalisation would be more effective in treating the depression than people whose beliefs reflected a primarily psychosocial model. Thus trainees' judgements of treatment effectiveness, for the same client, varied in relation to their own causal models. Causal attributions for the depression appeared to be congruent with trainees causal beliefs about mental illness as a whole. Further research would be helpful to understand how clinicians make these judgements, in real-life settings. This could explore the extent to which clinicians apply their own causal models to understanding and formulating client's distress and to what extent they use client data to create case specific models.

The finding that trainees who felt the depression was more likely to have biological causality were more likely to advocate anti-depressants, ECT and hospitalisation, may seem logical. However, there is no evidence to support this assertion (NICE, 2009). In fact, in the case of mild depression, the guidelines state, anti-depressants and medical treatments should not be considered due to a poor risk-benefit ratio. Of course, ratings of treatment effectiveness do not equate to actual endorsement or recommendation of medical treatments in clinical settings. Yet it seems feasible that clinicians who perceive anti-depressants to be more effective may be more likely to recommend them or support their use, even if not indicated by the evidence. Previous, studies have found this to be the case amongst medical professionals (Read & Harre, 2001). Future studies could test this hypothesis in relation to clinical psychologists.

There was no effect of causal beliefs on perceived effectiveness of psychological therapy. This suggests that trainees feel therapy is still effective even if the depression is seen to have biological causes. However, trainees who had high biological ratings felt the client would be less motivated and engaged in psychological therapy. In addition, there was some support that people who endorsed biological models perceived the client to be more risky, in terms of self-harm and need to be hospitalised. Previous, research has demonstrated that holding biological causal models can also lead professionals to have more blaming and authoritarian attitudes towards clients (Kent & Read, 1998; Miresco & Kirmayer, 2006).

It is unclear why biological models led to more pessimism about client engagement in therapy. It would be interesting to explore the impact of clinicians' casual beliefs on attitudes to real-life clients and whether clinical judgements impact the therapeutic relationship. Previous research has suggested that incongruence between clinicians' and clients' causal models of mental illness can lead to worse adherence and outcomes in therapy (Cottraux et al., 1993; Lax et al., 1992). Understanding how clinicians' models and client beliefs interact in therapy would be an interesting avenue for further research. Previous research suggests that biological explanations of depression can reduce service-users self-efficacy and hope for recovery, (Brown et al. 2007). If clinicians who hold strong biological models of mental illness are more likely to convey medical illness explanations to the client, in attempt to normalise symptoms, this may have inadvertent negative consequences for the client.

A tentative finding from this study was that trainees who did not strongly endorse either psychosocial or biological causal beliefs had lower perceptions of control, self-efficacy, and client engagement. Previous research has shown that not having a coherent causal explanation for emotional distress leads to reduced

perceptions of treatment effectiveness (Yopchick & Kim, 2009). A problem in making any inference from the current study is that the effect may be due to a bias in scoring, with participants in this group having a tendency to rate all measures in the lower range. One indication that this may not explain the whole effect is that there were no significant differences, across causal beliefs, in the check of motivation. If not having causal explanations has a reliable effect on clinical judgements this will be a significant finding and as such it warrants further research.

Limitations. There were a number of limitations of the current study. Firstly, the sample used trainee clinical psychologists and it is not clear if the findings would generalise to the wider population of qualified clinicians. Clinicians involved in the care of clients with depression routinely come from many different professional backgrounds such as nurses, psychotherapists, counselling psychologists, social workers and medical doctors. The use of clinical psychology trainees did not allow for comparisons to be made about the effect of aetiological labelling on clinical judgements across professional groups. This omission was primarily due to the large number of participants which this would have necessitated.

A second limitation, relates to the nature of the vignettes used. These adhered to the DSM diagnostic criteria for depression and clearly labelled the client's experiences as "depression." Thus the vignettes utilised diagnostic and medicalised language which may have primed participants towards an illness conceptualisation of the client's experiences. The use of diagnostic and medical language is common within mental health services. However, it would have been interesting to investigate whether a non-diagnostic presentation of the client's experiences would have interacted with the effects of aetiological labelling on clinicians' judgements. In addition, the use of vignettes does not enable the inference that the effects of causal beliefs on clinical

judgements would hold in real-life clinical settings. Clinical vignettes are widely used in research and do have validity; clinicians often receive written referral information about clients. Furthermore, the effect of heuristics and biases in clinical-decision making may actually be amplified in clinical settings due to time pressures.

A final consideration is the nature of the questionnaires used. These were reliant on self-report measures of attitudes rather than intentions or actual behaviour. The relationship between attitudes and behaviour is unlikely to be straight-forward. In addition, although conservative measures were adopted, the use of multiple comparisons means that caution needs to be taken in interpreting effects which were small or marginal.

Conclusion

This study offers preliminary support that holding biological causal models of depression can bias judgements of treatment effectiveness and client engagement, leading trainees to more strongly endorse medical treatments such anti-depressants and ECT, even though they are unlikely to be indicated by the evidence-base or client data. The effect sizes were substantive and warrant further confirmatory studies.

The experimental findings also suggested that labelling depression as biological can bias clinicians towards endorsing biological causal beliefs and medical treatments. This was a small effect but given the subtlety of the manipulation used in the study it seems worthy of further investigation.

There is a lack of research in the area of causal explanations and clinical judgements. The findings from this study can best seen as preliminary; further research would be valuable to set the findings in a more comprehensive context.

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Major Research Project

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Section C: Critical Appraisal

Word Count: 1960

What research skills have you learnt and developed from undertaking this project. What do you need to develop further?

Prior to clinical training I worked in purely clinical roles. As such I have not conducted research since my undergraduate degree, eight years ago. I was daunted by the major research project and the level of skill and knowledge it necessitated. One area of research I did feel confident in was critical appraisal. I think I was able to use and develop this skill in planning and developing my research idea.

I think the steepest learning curve for me was the recognition of how much time it took to plan and develop a research idea. I had little knowledge of the research process in practice and have a learnt a lot from doing this project. In hindsight, I think greater use of a systematic and time managed approach would have been beneficial. I think balancing multiple course, research, placement and home-life demands, as well looking for employment was exceptionally challenging and overwhelming at times. Although I hope never to have to balance so many competing demands again, I do feel I have a better appreciation of how to structure and plan research and could develop these skills further in clinical practice.

I think a key area of learning for me was the importance of a thorough literature search. I think initially my literature searching was somewhat haphazard and I found it difficult to funnel the process. I feel there was an element of pressure to get started with data collection prior to completing a thorough literature search. I definitely feel, over the course of the project, I significantly developed my literature reviewing skills. I think it would be useful to develop this skill further, so when I do research in the future it is well-founded in a research context prior to embarking on the project. I now have a better understanding of the crucial role of the literature search in the research process.

The biggest area of development for me was definitely in the advancement of my statistical knowledge. I have always had somewhat of an anxiety around statistics. I think this in part relates to my specific learning difficulties and in part to my undergraduate struggle with statistics. On reflection, it has made me query why I took on such a complicated quantitative project. Of course I was interested in the research and a quantitative analysis seemed the most appropriate research method. I also feel there was an element of me pushing and challenging myself to overcome my anxiety. Grappling with SPSS has definitely been challenging and time-consuming, particularly in the absence of research supervisors with quantitative expertise. I feel I have to some extent overcome my statistics demons, but I would like to develop these skills further. In particular, I feel I would like to gain knowledge of different types of analysis, such as multiple regression, and how to apply them to different research designs and questions. The knowledge I have developed will definitely be helpful in understanding and critiquing published quantitative research in the future.

A new experience for me was working with and collaborating with an external supervisor. This is something I have enjoyed and would like to have the opportunity to do in the future. I found the process supportive and have developed skills integrating and negotiating different perspectives and feedback in my work. I feel this experience will definitely be beneficial for when I do joint-working in the future.

If you were able to do this project again what would you do differently and why?

In a general sense, I would have tried to be more systematic and managed by time better. I feel I am someone who works well under pressure; however I think I

underestimated the amount of time the literature review, data-analysis, and reporting of the results would take. I think I also underestimated the impact of my dyslexia on the speed of my written work and my difficulties in concise writing and proof-reading. I think if I were to do the project again I would have afforded myself more time for these things.

I would definitely have spent more time exploring the literature prior to embarking on the study. The original idea for my study was based on my supervisors' unpublished study within a similar area, but related to anxiety. I realised quite late in the day that there was very little published research in the specific area of professionals' causal beliefs and clinical judgements in depression. I think if I had more time it would have been interesting to more thoroughly explore the clinical-decision making research and critical psychology literature, although this would have been beyond the scope of the written work.

Ideally, if I were to do this project again I would have sought more guidance and support with the data analysis. I have queried whether ANOVA was the most appropriate method for the exploratory analysis or whether regression analysis would have been better to examine the relationships between clinicians' causal beliefs and clinical judgements. I am very grateful for the support from the statistician at Canterbury Christ Church University, with some of the SPSS analysis. However, some more general guidance with regard to the design and analysis would have been valuable.

If I were to do this project again, I would have liked to explore clinician's perceptions of the client's blame and responsibility for the depression, as this is something that was highlighted in the literature (Miresco & Kirmayer, 2006). I also wonder if the perceived

stigma of others was the most appropriate measure of stigma or whether direct measures would have been more appropriate. However, using direct measures of stigma would need to overcome problems of social desirability.

I think a greater use of measures of hypothetical behavioural intentions would have added some validity to the hypothesis that causal bias, in judgements of treatment effectiveness and engagement, are likely to have real-life implications. For instance, these could have asked "if you were to treat the client how likely would you be to use (as examples): "medication-support," "illness models of normalisation", "recommend a psychiatric review/ CPN" or "give the client between-sessions homework?" Developing such a measure would have raised issues of validity and reliability, which would have needed to be overcome.

Finally, I would have spent more time exploring whether there are well-validated and established measures of clinicians' attitudes and therapy optimism available for use rather than relying on an amalgamation of two scales, which was likely to reduce the scale's validity. It may be that these scales are simply not available.

As a consequence of doing this study would you do anything differently in regard to clinical practice, or make clinical recommendations? If so, why?

I think this study has highlighted to me that how we frame emotional distress can have unexpected consequences. There is evidence that lay theories of emotional distress tend to reflect a psychosocial framework and that incongruence between clinician and client models may lead to worse outcomes and adherence for therapy (Cottraux, Messy, Marks, Mollard, & Bouvard, 1993; Lax, Basoglu and Marks, 1992). In addition, research

suggests that biomedical models of depression have been associated with less control over symptoms, lower self-efficacy and worse expected prognosis (Deacon & Baird, 2009; Nieuwsma & Pepper, 2010). As such, I think I will be far more cautious in using any illness analogies as a form of normalisation or psycho-education. I think it has also highlighted to me that I, and other clinicians, make judgements which are biased by our own beliefs about causality, and that this may lead us ignore client-specific data and/or the evidence-base. Although I feel clinical intuition is a useful clinical tool, I think clinicians need to remain reflective and be aware and explicit about their own causal models and potential biases. I think clinicians need to remain vigilant in considering clients in a person-centred, individual way, marrying this information with the clinician's expertise and the current evidence-based best practice.

This study has also emphasised to me the importance of clients' own causal models. I think in clinical situations it may be helpful to explore clients' causal models and develop these with the client to from a non-stigmatising and empowering part of the clients' formulation.

I think more generally it has highlighted the possible impact of labels, explanations, psycho-education and diagnosis. I think it has challenged me to be more critically aware of these issues.

If you were to undertake further research in this area what would that research project seek to answer and how would you go about it?

There appears to be very little research in the area of clinical-decision making and the impact of causal models on clinical judgements. As such there are many valuable

avenues for further research. Future studies could examine the extent to which causal models impact clinical-decision making in the light of other influences such as client-specific data (e.g. gender, recurrence of difficulties, specific symptoms/experiences, age etc.), evidence-based guidance, theoretical orientations and other potential moderating variables. A qualitative approach may be valuable due to the exploratory nature of such research. Clinical psychologists could be interviewed to explore their causal models and clinical judgements in relation to different client vignettes. Semi-structured interview techniques could be used to gain a comprehensive picture of how clinicians form client-specific causal models and the information that is important in making clinical judgements and planning treatments. I feel a grounded theory approach would be appropriate to develop a model of this process, which could then be tested through further quantitative studies.

A further interesting area of exploratory research would be to explore how clients' and clinicians' causal models interact in practice. Some previous research suggests that clinicians put more emphasis on biomedical causes than clients and that this can lead to different treatment preferences (Kuyken, Brewin, Power & Furnham, 1992). In turn, studies suggest that incongruence between treatment interventions and causal models can reduce motivation for treatment (Meyer & Garcia-Roberts, 2007). A study could examine the client's and clinician's model of the client's depression in practice and how congruency in models impacts treatment outcomes (such as success, drop-out, client satisfaction and acceptability). Such a study could implement a pre-post design collecting outcomes on reasons for depression, motivation for treatment and symptomology (pre- and post-treatment) and collect client and clinician feedback

on therapy process post-treatment. The data could be analysed using regression analysis to see if congruence does impact these treatment variables.

A number of predictions from my study would also warrant further testing. For example, the findings suggested that not holding causal model of the depression reduced perceptions of control over the depression and client self-efficacy. It is possible that this was a product of the measurement rather than a real effect. It would be interesting to examine this in a study which overcomes the scoring issues, either by using reverse-scored items or by using different types of measurement.

It would also be useful to replicate the exploratory analysis conducted in order to conduct planned comparisons and specific predictions as part of a robust research design. This research could explore clinicians' client-specific causal models of depression (using a vignette) without any experimental manipulation. The study could aim to overcome many of the limitations of the current study by using measures of blame and responsibility and more comprehensive, and client–specific, measures of clinicians' stigma and attitudes. Furthermore, this research could explore hypothetical behavioural intentions such as proposed treatments, number of sessions, specific strategies', likelihood of step-up or step-down referrals, and so on.

More generally, this study has made me interested in critical psychology ideas and specifically the impact and relevance of diagnostic labelling. I would be interested to explore these ideas further.

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Section D: Appendix

A. Search Methodology

Inclusion/Exclusion Criteria

Peer-reviewed studies were included if they measured biomedical causal beliefs, and/or illness beliefs, about depression. Studies which only described depressive symptoms, mood disorders or generic "mental illness" were excluded. Studies were included that examined public, service-users or professional attitudes. "Professionals" included people involved in the provision of treatments for, or management of, depression with relevant postgraduate qualifications such as psychotherapists, clinical psychologists, psychiatrists and social workers or trainees in a relevant profession. Studies which exclusively sampled from non-professional staff groups such as support workers were excluded from the review. Only English language articles were included.

Search Strategy

Searches were conducted (up to 2012) on the five electronic databases using the following key words:

> "mental disorders'' OR "mental illness'' OR "depression'' OR "depressive disorder" AND "attitudes'' OR "stigma" OR "treatment preferences'' OR "illness beliefs'' OR "illness attributions'' OR "causal beliefs" OR "biological beliefs" OR "medical beliefs" OR "psychological beliefs" OR "psychosocial beliefs''

Search terms were "auto-exploded" where possible, to include synonyms and other relevant terms. Search results were filtered based on the inclusion/exclusion criteria. Further studies were found by cross-checking reference lists for relevant articles.

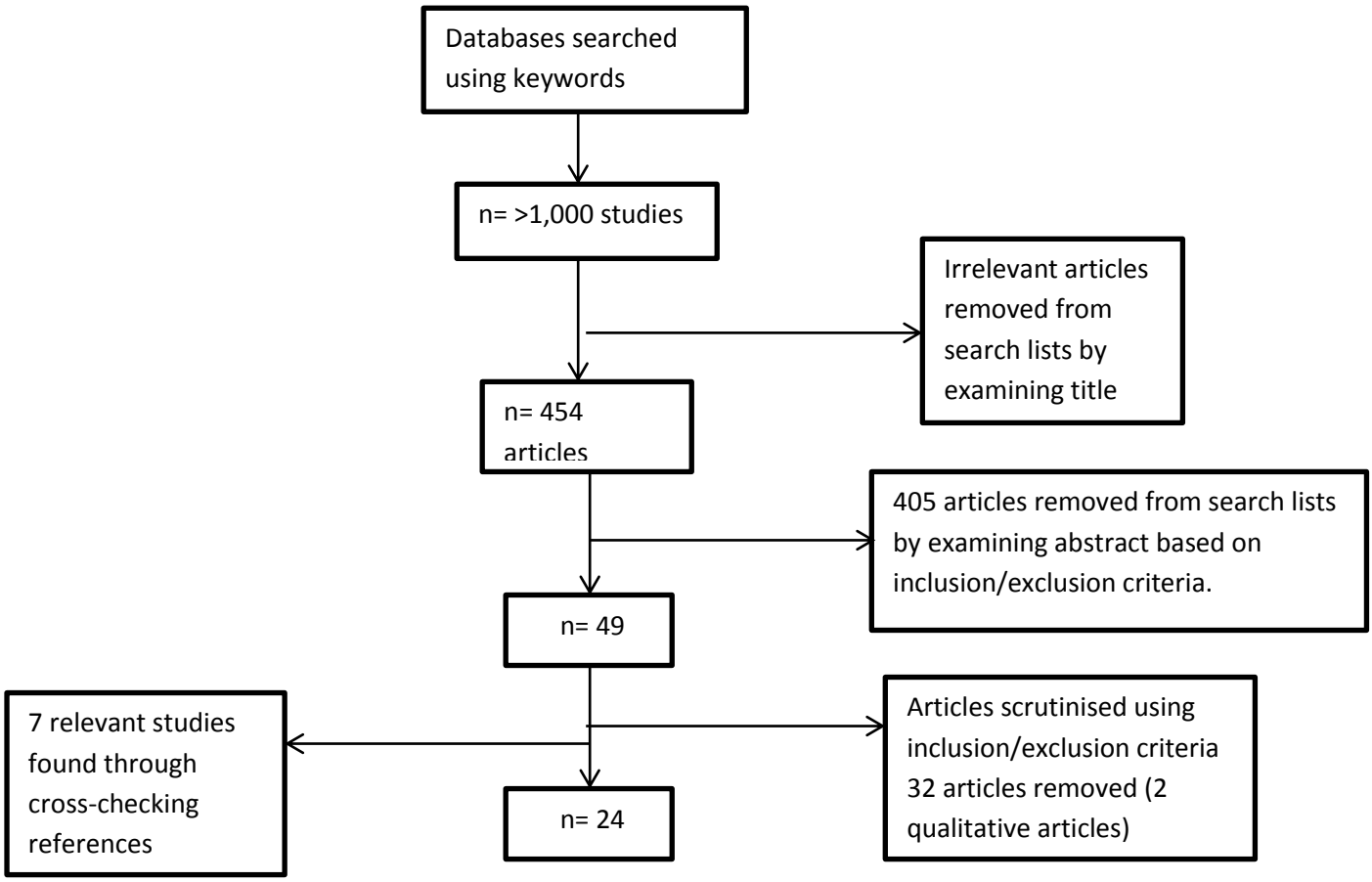


Figure 7.

Search Flowchart

B. Description of Studies

Twenty-four relevant studies were identified: population surveys (n= 9), observational/small-scale surveys (n= 11), and quasi-/experimental studies (n= 4). The studies reviewed were predominantly based in America (n= 11), but also included studies from: the UK (n= 5), Canada (n= 1), Australia (n= 2), Taiwan (n= 1), Holland (n= 1), Sweden (n= 1), Switzerland (n= 1) and Ireland (n= 1). Populations sampled included lay (n= 10); depressed service users (n= 9), professionals (n= 1) and mixed (n= 2).

Authors	N	Design/ Analysis	Inclusion criteria/ sample	Constructs measured	Measures	Most common EBs	BM beliefs (% of sample)	Biomedical ABs associated with negative attitudes to depression?	ABs: Impact on treatment and help-seeking
Ahn et al. (2009)	Study 1: P= 30/CP= 30/SW= 29; Age/f= NR Study 2: P=16/CP =25/SW= 22; Age= µ= 52.4 f= 29 Study 3: P= 8/CP= 16/SW= 20 Ageµ= 54 f= 22	Survey/Quasi-experimental (New Haven/ America) ANOVA	NA	ABs TE	NA-author designed	PS/BM	NA	NA	BM associated with higher belief in medication effectiveness** and lower belief in therapy effectiveness**
Brown et al. (2007)	191 f= 135 Age= µ= 45.1	Survey (Pittsburgh, USA) Regression Analysis	a.) ≥18 years b.)current research participants c.)Receiving anti-depressant treatment	ABs Control Consequences Duration Functioning Coping	Illness Perception Questionnaire (Weinman, 1996) SF6 health scale (Ware et al., 1994) The MOS health survey (Stewart et al., 1988). Brief COPE (Carver, 1997).	PS	NR	**Yes** – Less control over symptoms of D (*r*= .17*) **Yes** – Greater severity of consequences (*r*= .24*)	NA
Budd et al. (2008)	194 f= 108 Age= µ= 45	Survey (Wales) Regression Analysis	a.) Members of the Depression Alliance Cymru (Support group)	AB'S TE	NA- Author designed	PS/BM	NR	NA	Chemical-imbalance associated with greater TE for medication (*r*= .2**)

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

Deacon & Baird (2009)	90 F= 88.9% Age= 18-29 (μ= 20.9)	Quasi-experimental (Wyoming, America) *t*-tests	a.) Enrolled in abnormal psychology classes	ABs Self-stigma Credibility Public stigma Prognosis TE	Perceptions of depression questionnaire (PDQ; Deacon & Baird, 2009)	PS	20%	**Yes** – worse prognosis (*d*= .51**) and less self-efficacy (*d*= 1.76**) **No**- less self-blame (*d*= 1.01**), less perceived stigma (*d*= 1.20**), less belief in personal weakness (*d*= .65**)	Chemical-imbalance and greater perceived TE for medication (*d*= 1.20**) and greater perceived TE for psychotherapy (*d*= 1.12**)
France et al. (2007)	262 F= 59.5% Age= 18-58 (μ= 23.6)	Convenience survey (Midwest America) ANOVA	a.)≥18 b.)Enrolled on psychology classes	Help-seeking ABs	NA – author designed	PS/BM	Chemical-imbalance = 84.7%	NA	Endorsement of chemical-imbalance associated with stronger preference for medical help* and less preference for talking therapies*.
Goldstein & Rossellli (2003)	66 f= 44 Age= 18-22	Survey (Connecticut, USA) Regression Analysis	a.) University students	ABs Empowerment TE Stigma	NA-author designed	BM	NR	**No –** reduced blame(*r*= .32*), increased help-seeking (*r*= .30*), positive attitudes (*r*= .25*)	BM and greater perceived TE for psychotherapy (*r*= .29*)
Han et al. (2006)	N= 299 f= 218 Age= μ= 20.3	Experimental (Taiwan) ANCOVA	a.) University undergraduates	Biological ABs Blame Help-seeking	Author designed: Biological Attribution Scale, Psychological Blame Scale, Help-Seeking Willingness Scale	NA	NR	NR	BM education increased BM ABs** BM education was associated with increased help-seeking willingness*

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

Hansson et al. (2010)	319 F= 72.9% Age= 18-69 (μ= 43.8) *t*-tests	Survey (Sweden)	a.)Primary care patients b.)Recent depressive episode	ABs	NA– author designed	PS	3.6%	NA	NA
Jorm et al. (1997)	N= 2031 f= NR Age= NR	Household survey (Australia) x^2	a.) Private residents b.)Age= 18-74 years	ABs Risk factors Recognition	NA	PS	50% genetic causes	NA	NA
Jorm & Griffiths (2008)	3998	Household survey (Australia) Regression analysis	a.) ≥18 years	Stigma: Social distance Dangerous	Social distance scale (Link et al. 1999) Depression Stigma Scale (Griffiths et al. 2006;2008)	NR	NR	**Trend**-dangerousness OR 0.98 (*p*>.98) **Trend -** social distance (*p*>.06)	NA
Khasla et al. (2011)	145 F= 59% Age= 18-70	Observational (Philadelphia, America) Regression analysis	a.)Major Depressive Disorder b.)No comorbidity	Treatment preference ABs	RFD Addis et al., 1995).	PS	NR	NA	Trend to strong belief in chemical-imbalance and preference for medication *(d*= .32; *p*<.07). PS beliefs associated with preference for psychotherapy (*d*= .47**)
Kuyken et al. (1992)	SU= 20 CP= 25 L= 49 Age/ gender: NR	Interview/ survey (London, UK) ANOVA	NA	ABs Credibility TE	NA– author designed	L= PS CPs/SUs = BM	SUs= 65% CPs= 48% L= 14%	NA	Stronger BM beliefs associated with increased perceived TE for medication**

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

Lauber et al. (2003)	873 F= NR Age= NR	Telephone survey (Switzerlan) NA	NA	EBs	NA– author designed	PS	Illness beliefs= 25.7%	NA	NA
Leykin et al. (2007)	N= 172 f= 101 Age= μ= 40.4	Observationl (Pennsylvania, America) ANCOVA	a.)RCT participants for CT efficacy b.)≥18years c.)Major Depression	EBs Depressive symptomology	RFD (Addis et al., 1995) Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960).	PS	NR	NA	Trend towards higher BM beliefs and worse outcomes in CT (p<.09) Successful treatment reduces treatment incongruent ABs: CT(d= -.36*) ADs (d= -.54**)
Meyer & Garcia-Roberts (2007)	N= 97 f= 61 Age= μ= 39.21	Survey (London, UK) Regression analysis	a.)Receiving psychological therapy for depression in primary care setting	ABs Motivation	RFD (Addis et al. 1995). Author developed: "Motivations For Interventions" scale (MFI).	PS	NR	NA	BM beliefs correlate with motivation for medical treatments (r= .77**)
McKeon & Carrick (1991)	1403 f= 701 Age= NR	Survey (Ireland) Descriptive	a.) ≥16years	Stigma Treatability Causes Treatment preference Illness belief.	NA	PS	Illness beliefs = 33% Chemical-imbalance = 9%	NA	NA

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

Nieuwsma & Pepper (2010)	N= 69 f= NR Age= NR	Observational (Wyoming, America) Regression analysis	a.) Undergraduate psychology students	ABs Stigma Self-efficacy	Modified - Survey of etiological beliefs and treatment effectiveness (Goldstein & Rosselli, 2003) Stigma Scale (Nieuwsma & Pepper, 2010) PDQ– Self efficacy subscale (Manber et al., 2003)	PS/BM	NR	**No** - no significant association between BM ABs and stigma. **No** - PS ABs correlated with higher authoritarian stigma *r*= .29*	PS ABs correlated with perceived TE of self-initiated treatments (*r*= .33**). **Trend** - BM ABs correlated with perceived TE of medical treatment (*r*= .23, *p*<.06)
Ogden et al. (1998)	N= 769 <u>Patient:</u> n= 681/ f= 481 Age= μ= 44.81 <u>GPs:</u> n= 90/ f= 33 Age μ = 41.92	Survey (England) ANOVA	a.)≥16years b.)Patients or GPs from nine practices in England	ABs	NA	L= PS GPs= BM/PS SUs= BM/PS	NR	NA	NR
Paykel et al. (1998)	N= 1991: 2009 1995:2050 1997:1946 f= NR Age= NR	Household survey (Great Britain) ANOVA	a.)≥15 years b.)Residents	ABs Illness belief Stigma Willingness to seek help	NA	PS	In 1997: Medical illness belief = 81% Brain changes = 43%	NA- Stigmatising attitudes remained consistent over the time period.	Willingness to seek professional support and endorsement of anti-depressant treatment increased over time in line with increased illness beliefs.

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

Pescosolido et al. (2010)	N= 1956 f= 998 Age= μ= 45	Household Survey (America) Logistical regression	a.)Residents b.)≥18 years	ABs TE Stigma	NR	NR	Chemical-imbalance = 80	**Yes -** dangerousness to self OR= 5.04** dangerousness to others OR= 2.70**	NA
Rusch et al. (2009)	N= 86 f= 62 Age μ= 21.45	Experimental (Wisconsin, America) ANOVA	a.) Undergraduate psychology students	ABs Stigma Behavioural intentions	Depression beliefs (Goldstein & Rosselli, 2003) Depression Attribution Questionnaire-27 (Corrigan et al., 2003).	PS/BM	NR	**NA**- increasing biological attribution did not significantly reduce stigma.	NA
Schweizer et al. (2010)	221 F= 128 Age: μ= 42	Observational Community clinic (Maastricht, Holland) ANOVA	a.)Major depression/dysthymia b.)No acute suicide risk	ABs Treatment preference	Reasons For Depression (RFD; Addis, Taux & Jacobson, 1995).	PS	NR	NA	BM ABs associated with preference for medication* PS ABs associated with preference for CBT*
Srinivasan et al. (2003)	N= 102 f= 67 Age= μ= 41.1	Out-patient survey (Canada) Regression analysis.	a.) Depressive disorder b.)Referred to a psychiatric outpatient clinic	ABs	NA-author designed	PS	NR	NA	NA
Wong et al. (2010)	N= 223 f= 156 Age= μ= 23.6	Survey/ Content analysis (USA) x^2	a.)>18 years b.)East, South, South East Asian decent c.) Psychology graduates/ therapists excluded	ABs Willingness to seek help Enculturation	Asian American Values Scale – Multidimensional (Kim et al., 2005).	PS	BM= 25.1.	NA	BM ABs associated with greater willingness to seek professional help**

AB= Aetiological belief; AD= Anti-depressant; BM= Biomedical; CT= Cognitive Therapy; CP= Clinical Psychologist; D= Depression, GP= General Practitioner; NA= Not applicable; NR= Not reported; P= Psychiatrist; PS= Psychosocial; TE= Treatment Effectiveness; RCT= Randomised Control Trial; SW= Social Worker; *p<0.5; **p<0.01

D. Vignettes

Biological condition

D is experiencing difficulties typical of biological depression including depressed mood, poor sleep, reduced appetite and poor motivation. D has been struggling with these symptoms for almost six months and this is significantly impacting their functioning.

D reports difficulty sleeping and has been experiencing early morning waking. D rarely sleeps in past 4 or 5am. As a result they spend long periods of time lying in bed ruminating about their problems. They have also noticed a reduction in their appetite and frequently skip meals.

They have been feeling very lethargic and lacking in energy. When not at work D rarely feels up to doing much. D used to like going for a run before work but despite waking early no longer has the drive to do so.

D has a close family. D's cousin had a significant episode of depression for which they had treatment. D's family were very supportive and have a good understanding of the impact of depression. However, D finds it very difficult to talk about how they feel and has become withdrawn from both family and friends.

D has always enjoyed work but is struggling to stay motivated and concentrate and their performance is beginning to slide. Their reduced performance has been highlighted in a recent appraisal and D is beginning to worry about their own competence. D is often tearful and feels guilty about their poor work performance and for ignoring friends.

D sometimes feels life is not worthwhile anymore and wishes they weren't here. Although finding it difficult to cope D has not expressed any suicidal intention or thoughts of harming themself.

(Respondents: 33.34%)

Psychosocial condition

D is experiencing difficulties typical of psychosocial depression including depressed mood, poor sleep, reduced appetite and poor motivation. D has been struggling with these symptoms for almost six months and this is significantly impacting on their functioning.

D is spending long periods of time lying in bed ruminating about their problems. As a result they have had difficulty sleeping and have been waking early in the morning, rarely sleeping in later than 4 or 5am. They have also noticed a reduction in their appetite and frequently skip meals.

They have been feeling very lethargic and lacking in energy. When not at work D rarely feels up to doing much. D used to like going for a run before work but despite waking early no longer has the drive to do so.
D has a close family. D's cousin had a significant episode of depression for which they had treatment. D's family were very supportive and have a good understanding of the

impact of depression. However, D finds it very difficult to talk about how they feel and has become withdrawn from both family and friends.

D has always enjoyed work but is struggling to stay motivated and concentrate and their performance is beginning to slide. Their reduced performance has been highlighted in a recent appraisal and D is beginning to worry about their own competence. D is often tearful and feels guilty about their poor work performance and for ignoring friends.

D sometimes feels life is not worthwhile anymore and wishes they weren't here. Although finding it difficult to cope D has not expressed any suicidal intention or thoughts of harming themself.

(Respondents: 33.33%)

Control (neutral) condition

D is experiencing difficulties typical of depression including depressed mood, poor sleep, reduced appetite and poor motivation. D has been struggling with these symptoms for almost six months and this is significantly impacting their functioning.

D is spending long periods of time lying in bed ruminating about their problems. As a result they have had difficulty sleeping and have been waking early in the morning, rarely sleeping in later than 4 or 5am. They have also noticed a reduction in their appetite and frequently skip meals.

They have been feeling very lethargic and lacking in energy. When not at work D rarely feels up to doing much. D used to like going for a run before work but despite waking early no longer has the drive to do so.

D has a close family. D's cousin had a significant episode of depression for which they had treatment. D's family were very supportive and have a good understanding of the impact of depression. However, D finds it very difficult to talk about how they feel and has become withdrawn from both family and friends.

D has always enjoyed work but is struggling to stay motivated and concentrate and their performance is beginning to slide. Their reduced performance has been highlighted in a recent appraisal and D is beginning to worry about their own competence. D is often tearful and feels guilty about their poor work performance and for ignoring friends.

D sometimes feels life is not worthwhile anymore and wishes they weren't here. Although finding it difficult to cope D has not expressed any suicidal intention or thoughts of harming themself.

(Respondents: 33.33%)

E. Survey

Instructions

Following completion of the consent form you will be redirected to a client vignette. Please take time to read the vignette. Following this a series of questionnaires will be presented. These ask questions relating to your beliefs and thoughts regarding the client.

The questionnaire should take no longer than 15-20 minutes to complete. You can leave the survey at any point by pressing the exit survey link in the top right hand corner of the screen. If you choose to leave the survey it will be assumed you have used your right to withdraw study and your answers will not be included in the research. Once you have completed the questionnaire you will be asked to fill in your demographic details.

IF YOU WISH TO ENTER THE PRIZE DRAW YOU WILL NEED TO CLICK ON A LINK TO FILL IN A REGISTRATION FORM. THIS ASKS FOR YOUR NAME AND EMAIL ADDRESS TO ENABLE US TO CONTACT YOU IF YOU WIN. THIS INFORMATION WILL NOT BE LINKED TO YOUR DATA OR USED FOR ANY OTHER PURPOSE. THERE ARE 4 PRIZES OF AMAZON GIFT VOUCHERS WORTH £50, £25, £15 AND £10. THE DRAW WILL TAKE PLACE ONCE DATA COLLECTION HAS BEEN COMPLETED.*

If you wish to receive a study debrief or have any queries please contact myself using the contact details emailed to you, which will also be presented again at the end of the survey.

*no later than March 2013

Consent

THANK YOU FOR YOUR PARTICIPATION

To continue, please read the following statements and check the box to confirm your agreement:

1. I agree to participate in this study and to complete an online questionnaire for the purposes of the study described.

2. I understand that my participation is voluntary and I am free to withdraw at any time before the completion of data collection.

3. I understand that data will be kept confidentially and securely and will be anonymised for write up.

4. I understand that to maintain your anonymity a full debrief about the purpose of the study will not be provided automatically. This debrief can be requested by emailing the researcher on the contact details provided.

5. I understand if I have any questions or concerns I can contact the researcher using the contact details given.

[ ] I CONFIRM I AGREE WITH THE STATEMENTS LISTED

Vignette

Please read the following description of Client D's depression. You will then be asked to answer a series of questions. These will involve you reflecting upon your thoughts and ideas relating to this client.

[*Random presentation of vignette*]

Motivation check

Please check the symptoms of depression that client D is experiencing.

□ Low or depressed mood
□ Difficulties sleeping or sleeping too much
□ Loss of interest or pleasure
□ Feelings of worthlessness
□ Difficulties concentrating
□ Loss of energy
□ Guilt
□ Irritability
□ Changes in appetite
□ Thoughts of harming oneself or/and thoughts of suicide

Causal beliefs

Holding the client in mind, please indicate how much you feel each of the following factors is likely to be a leading cause in their depression.

	Definitely not a cause 1	Slightly not a cause 2	No opinion 3	Slightly a cause 4	Definitely a cause 5
Psychological factors[PS]	□	□	□	□	□
Biological factors [B]	□	□	□	□	□
Environmental factors[PS]	□	□	□	□	□
A chemical imbalance in the brain or nervous system [B]	□	□	□	□	□
Lack of social support[PS]	□	□	□	□	□
Learned helplessness[PS]	□	□	□	□	□
A genetic or inherited predisposition [B]	□	□	□	□	□
Biochemical abnormalities [B]	□	□	□	□	□
Recent misfortunes[PS]	□	□	□	□	□
General stress[PS]	□	□	□	□	□
Response to a negative life event[PS]	□	□	□	□	□
Disease in the brain [B]	□	□	□	□	□
Expecting too much of one's self[PS]	□	□	□	□	□
A poor cognitive outlook on the world[PS]	□	□	□	□	□
Biological changes within the brain or nervous system [B]	□	□	□	□	□

A melancholic personality[PS]	☐	☐	☐	☐	☐
Lack of will power[PS]	☐	☐	☐	☐	☐

NB. [PS] denotes items on the psychosocial scale, [B] denotes items on the biological scale

Treatment effectiveness

How effective do you feel the following would be in treating this client's depression?

	Definitely Ineffective 1	Moderately Ineffective 2	Slightly Ineffective 3	Not sure/ possibly 4	Slightly Effective 5	Moderately Effective 6	Definitely Effective 7
Antidepressants[M]	☐	☐	☐	☐	☐	☐	☐
Hospitalisation[M]	☐	☐	☐	☐	☐	☐	☐
Electro-convulsive therapy (ECT) [M]	☐	☐	☐	☐	☐	☐	☐
Vitamins/herbal remedies[SM]	☐	☐	☐	☐	☐	☐	☐
Recreational Drugs[SM]	☐	☐	☐	☐	☐	☐	☐
Alcohol[SM]	☐	☐	☐	☐	☐	☐	☐
Psychotherapy[P]	☐	☐	☐	☐	☐	☐	☐
Behavioral Therapy[P]	☐	☐	☐	☐	☐	☐	☐
Cognitive Therapy[P]	☐	☐	☐	☐	☐	☐	☐
Deal with it alone[SI]	☐	☐	☐	☐	☐	☐	☐
Exercise[SI]	☐	☐	☐	☐	☐	☐	☐
Relaxation / meditation / yoga[SI]	☐	☐	☐	☐	☐	☐	☐
Psychoeducation[SI]	☐	☐	☐	☐	☐	☐	☐
Getting out more[SI]	☐	☐	☐	☐	☐	☐	☐

NB. [M] denotes items on the medical subscale, [P] denotes items on the psychological therapy subscale, [SM] denotes items on self-medication subscale, [SI] denotes items on the self-initiated subscale

Self-efficacy

To what extent do you feel the client would be able to use the following to manage their depression?

	Not at all 1	Somewhat 2	Quite a lot 3	Very much so 4
Clarifying their priorities in life	☐	☐	☐	☐
Improving their relationships with others	☐	☐	☐	☐
Understanding themselves better	☐	☐	☐	☐
Increasing their social support	☐	☐	☐	☐
Changing how they think about themself	☐	☐	☐	☐
Changing their behaviours	☐	☐	☐	☐
Working hard at solving some of their problems	☐	☐	☐	☐
Having a confidant	☐	☐	☐	☐
Seeking help from talking to others	☐	☐	☐	☐
Making changes in their life	☐	☐	☐	☐
Improving their family situation	☐	☐	☐	☐

Letting time heal	☐	☐	☐	☐
Increasing their activities	☐	☐	☐	☐
Getting psychotherapy	☐	☐	☐	☐
Learning to cope with stress	☐	☐	☐	☐
Exercising regularly	☐	☐	☐	☐
Improving their diet	☐	☐	☐	☐
improving their health	☐	☐	☐	☐
Developing a relationship	☐	☐	☐	☐
Becoming more centred/well-balanced	☐	☐	☐	☐
Using self-help	☐	☐	☐	☐
Having an explanation for their depression	☐	☐	☐	☐
Participating in support groups	☐	☐	☐	☐
Making changes in their situation	☐	☐	☐	☐

Control-cure

Holding the client in mind, please indicate how much you agree with the following statements.

	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree
	1	2	3	4	5
Their depression will improve with time	☐	☐	☐	☐	☐
Recovery from their depression is largely based on chance[C]	☐	☐	☐	☐	☐
Treatment will be effective in curing their depression	☐	☐	☐	☐	☐
There is a lot they can do to control their depression[C]	☐	☐	☐	☐	☐
What they do determines whether their depression gets better or worse[C]	☐	☐	☐	☐	☐
There is very little that can be done to improve their depression	☐	☐	☐	☐	☐

NB. [c] denotes items on the control subscale

Stigma

For the following statements, think of how other people might treat this client. Use the scale provided to rate your agreement with each statement.

In general, other people would…

	Strongly disagree	Disagree	Neither agree nor disagree	Agree	Strongly agree
	1	2	3	4	5
…perceive them as unpredictable[Dg]	☐	☐	☐	☐	☐
…think they are incompetent[A]	☐	☐	☐	☐	☐
…discriminate against them[D]	☐	☐	☐	☐	☐
…see them as needy[Dp]	☐	☐	☐	☐	☐

…pity them[Af]	☐	☐	☐	☐	☐
…try to control them[A]	☐	☐	☐	☐	☐
…treat them unfairly[D]	☐	☐	☐	☐	☐
… not think they have self-control[Dg]	☐	☐	☐	☐	☐
…become angry with them[Af]	☐	☐	☐	☐	☐
…view them as irresponsible[A]	☐	☐	☐	☐	☐
…believe they are dependent[Dp]	☐	☐	☐	☐	☐
… not give them equal treatment[D]	☐	☐	☐	☐	☐
…believe they are aggressive[Dg]	☐	☐	☐	☐	☐
…get annoyed with them[Af]	☐	☐	☐	☐	☐
…believe that they need additional care[A]	☐	☐	☐	☐	☐
…be frightened by them[Dg]	☐	☐	☐	☐	☐
…ridicule them[Af]	☐	☐	☐	☐	☐
…be biased against them[D]	☐	☐	☐	☐	☐
…be insecure when with them[Af]	☐	☐	☐	☐	☐
…attempt to make decisions for them[A]	☐	☐	☐	☐	☐
…think they are dangerous[Dg]	☐	☐	☐	☐	☐
…be irritated by them[Af]	☐	☐	☐	☐	☐
…would see them as powerless[A]	☐	☐	☐	☐	☐
…would be unjust towards them[D]	☐	☐	☐	☐	☐
… would become frustrated with them[Af]	☐	☐	☐	☐	☐

NB. [Dg] denotes dangerousness, [A] denotes authoritarianism, [D] denotes discrimination, [Dp] denotes dependency, [Af] denotes affectivity.

Clinicians' attitudes

If you, or a psychologist within an appropriate service, were able to offer this client psychological therapy…

How motivated do you feel the client would be for treatment?
Where 0% is completely unmotivated and 100% is completely motivated.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How effective do you feel treatment would be?
Where 0% is completely ineffective and 100% is completely effective.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

If treatment was successful how likely do you feel it is that that they would relapse following treatment?

Where 0% is that they are definitely going to remain well and 100% is that they will definitely relapse.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How intensive do you feel therapy would need to be?
Where 0% is the least intensive available and 100% is the most intensive available.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How likely do you feel they would be to drop out of treatment?
Where 0% is that they would definitely complete treatment and 100% is that they would definitely drop out of treatment.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How curable do you think their depression is?
Where 0% is completely incurable and 100% is completely curable.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Is hospitalisation needed?
Where 0% is hospitalisation is definitely not needed and 100% is that hospitalisation is definitely needed.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How disabling do you think their depression is for them?
Where 0% is not at all disabling and 100% is completely disabling.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

How likely do you think they would be to harm themselves?
Where 0% means that you feel they would definitely not harm themself and 100% is that they will definitely harm themself.

0%	10%	20%	30%	40%	50%	60%	70%	80%	90%	100%

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Demographics and background information

Thank you for answering the questions in relation to the client vignette. Please spend a few more moments completing the demographic and background questionnaire.

What do you think was the purpose of the study?

[*free response*]

What is your gender?

Male Female

What is your age?

[*drop down menu to enter age*]

What is your ethnicity?

Black British	☐
Black African	☐
Black Carribean	☐
Black other	☐
White British	☐
White European	☐
White other	☐
Pakistani	☐
Indian	☐
Chinese	☐
Asian other	☐
Mixed race	☐
Other	☐
Prefer not to say	☐

If other, please state: [*free response*]

What year of clinical training are you in?

First	Second	Third
☐	☐	☐

Which clinical training course on you on?

_Bangor University North Wales

_University of Bath
_University of Birmingham
_Coventry and Warwick
_University of East Anglia
_University of East London
_University of Edinburgh NHS Scotland
_University of Essex Tavistock
_University of Exeter
_University of Glasgow NHS Scotland
_University of Hertfordshire
_Institute of Psychiatry, King's College London
_Lancaster University
_University of Leeds
_University of Leicester
_University of Liverpool
_University of Manchester
_Newcastle University
_North Thames University College London
_Oxford
_Plymouth University
_Royal Holloway, University of London
_Salomons, Canterbury Christ Church University
_University of Sheffield
_Shropshire and Staffordshire
_University of Southampton
_South Wales
_University of Surrey
_Teesside University
_Trent Universities of Lincoln and Nottingham

Would you identify yourself with a particular theoretical orientation?

Yes □
No □
Not sure □

If yes, please state [*free response*]

Please rate your agreement with the following statements using the scale provided.

	Strongly disagree 1	Mildly disagree 2	Neutral 3	Mildly agree 4	Strongly agree 5
Psychology is the most important factor in the cause of mental illness.	□	□	□	□	□
Biology is the most important factor in the cause of mental illness.	□	□	□	□	□
The environment is the most important factor in the cause of mental illness.	□	□	□	□	□

Closing information

Thank you for your time and participation with this research study. You can request a full debrief and information about the study by email. If you would like to do this or have any questions regarding the study please contact the principal researcher on the contact details below. If requested, debrief information will be sent out following completion of the study.

If you are concerned that you may need support around your own emotional wellbeing please contact your GP or for advice about yourself or someone else you can call NHS direct on 0845 4647 or visit: www.nhsdirect.nhs.uk .

For advice and information on depression you can also go to:
http://www.nhs.uk/Conditions/Depression/Pages/Introduction.aspx

Contact details:
Kerry Tate
Trainee Clinical Psychologist
Salomons Campus, Canterbury Christchurch University
Email: kt149@canterbury.ac.uk
Supervised by:
Dr Blake Stobie
Consultant Clinical Psychologist
Centre for Anxiety Disorders and Trauma .
Email: blake.stobie@kcl.ac.uk

Professor Paul Camic
Research Director
Canterbury Christchurch University
Email: paul.camic@canterbury.ac.uk

If you want to make a complaint about the research:
Please direct any complaints to Prof Margie Callanan,
Department of Applied Psychology, Canterbury Christ Church University, Broomhill Rd., Tunbridge Wells, Kent TN3 0T

To enter the prize draw please add your contact details by following the link Click here to take survey

F. Factor Analysis

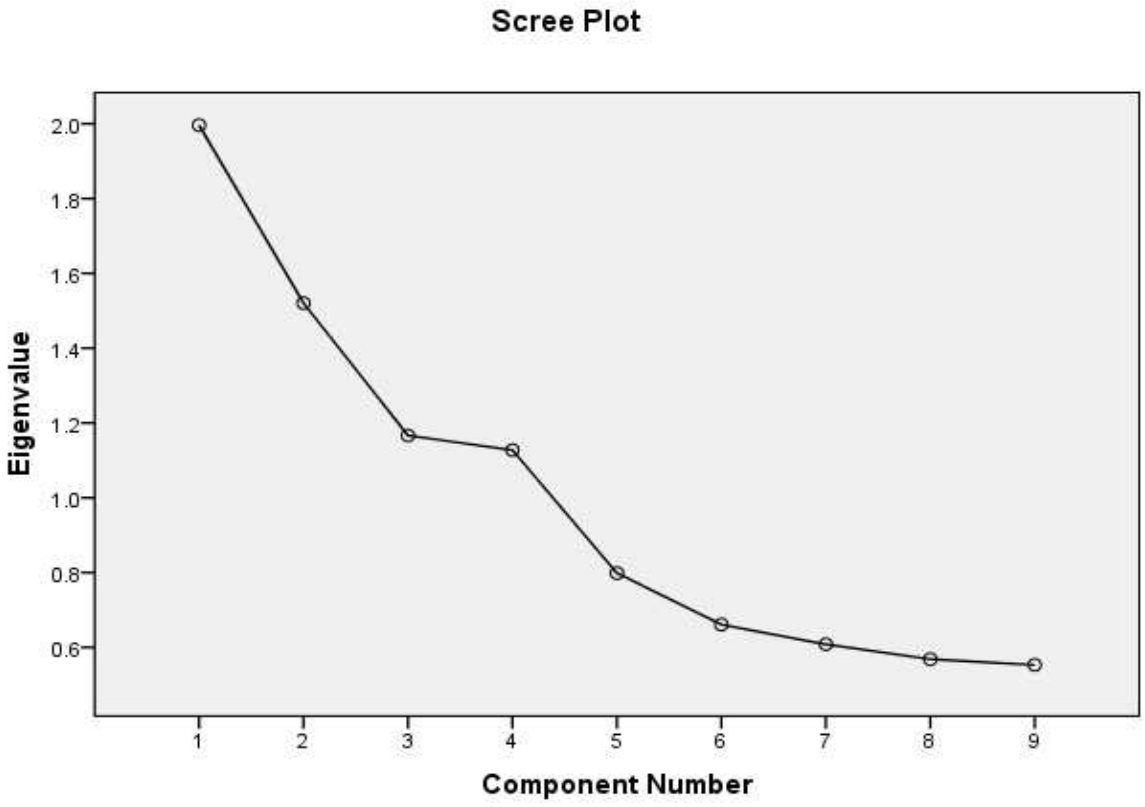
Table of factor analysis item loadings

	Engagement	Treatment effectiveness	Severity	Risk
Motivation	.77*	.07	-.13	-.30
Drop out	.74*	.09	-.11	.21
Relapse	.57*	.06	-.07	.37
Curability	-.06	.87*	.05	.06
Effectiveness	.24	.78*	-.12	-.01
Disability	.06	.02	.79*	-.34
Intensity of treatment	-.13	-.08	.72*	.26

needed				
Risk of self-harm	.38	-.07	.41	.48*
Need for hospitalisation	.06	.08	.04	.82*

*Designates the highest loadings.

Scree plot (spss output)



G. Ethics Approval

THIS HAS BEEN REMOVED FROM THE ELECTRONIC COPY

H. Recruitment Email

Dear XXXX,

I am a second year trainee from Salomons Clinical Doctorate course. I am currently recruiting participants for my thesis. The study has full ethics approval (a copy is attached to the email.

I would be very grateful if you could forward this email to all trainees on the XXXX Clinical Psychology course.

Thank you

XXXX

Dear Trainee

I am currently recruiting participants to take part in my major research project. The research has received full ethics approval from Salomons Ethics Panel.

As a thank you for your time you can enter into a prize draw to win one of 4 Amazon vouchers for £10, £15, £25 or £50.

The study involves completing an anonymous survey examining clinicians' beliefs about mental health problems. This should take no longer than 15-20 minutes to complete.

Please see attached information sheet for further details and contact information.

To participate please click on the link below:
https://www.surveymonkey.com/s/896VDPZ

Thank you

XXXX

Department of Applied Psychology
Canterbury Christ Church University
Salomons Campus
Broomhill Road
Tunbridge Wells, Kent TN3 0TG

I. Participant Information Sheet:

Canterbury Christ Church University

Salomons Campus at Tunbridge Wells,

Department of Applied Psychology

Faculty of Social and Applied Sciences

Participant Information Sheet

This study is part of a third year major research project for the Clinical Psychology Doctorate at Canterbury Christ Church University.

What is the study about?

The study is looking at clinicians' beliefs about mental health problems. A full debrief about the purpose and rationale for the study can be requested from the researcher. This will be emailed once data collection has been completed.

Why have I been asked to take part?

As a clinical psychology trainee your perspectives are valued for the purposes of this study.

Will I be paid to take part?

No, however everyone who takes part will be able to entered into a free prize draw. There are 4 prizes of Amazon vouchers worth £50, £25, £15, £10.

Do I have to take part?

No. Participation is voluntary. If you decide to take part you are still free to withdraw at anytime and without giving a reason.

What will happen if I do agree to take part?

1. If you agree to take part you can access the survey via the hyperlink. You will be asked to consent to your participation in the study. You are free to withdraw your consent up until the point of completing the survey.

2. If you agree to take part in the study, you will be presented with a set of instructions and asked to read a vignette.

3. You will then be asked to complete a series of questionnaires. This should take no more than 15-20 minutes.

4. Once you have completed the survey you will be able to access a prize draw registration form. You will be contacted by email if you win. Personal details for the prize draw will be kept separately from the research data maintaining anonymity. The prize draw will be completed within 2 weeks of data completion (no later than March 2013)

5. If you would like to receive further information about the study or a full debrief you can email the researcher to request this. Once the entire questionnaire data has been collected you will then be emailed with a full debrief.

Will my taking part be kept confidential?

Information will be kept confidential and your name and identifying data will NOT appear in any reports.

What will happen to the results?

Results will be written up for the purposes of a doctorate thesis and submitted for publication.

Contact details:

Should you have any further queries, or would like further information, please contact Kerry Tate who will be carrying out the research.

Confidentiality

All data and personal information will be stored securely within Canterbury Christ Church University premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by the principal researcher XXXX and research supervisors (XXXXX).

After completion of the study, all data will be remain anonymous.

[CANDIATE INFORMATION REMOVED]

If I want to make a complaint about the research.
Please direct any complaints to Prof Margie Callanan,
Department of Applied Psychology, Canterbury Christ Church University, Broomhill Rd., Tunbridge Wells, Kent TN3 0TG

J. Research summary for participants

Canterbury Christ Church University

Salomons Campus at Tunbridge Wells,
Department of Applied Psychology
Faculty of Social and Applied Sciences

Re. Debrief and summary: Clinician's attitudes study

Thank you for taking the time to participate in the on-line study exploring clinicians' beliefs about depression. All the data has now been collected and the study has been submitted as part of my clinical doctoral thesis.

The study

Over 200 trainee clinical psychologists, across England, Scotland and Wales, took part in the survey. Participants were randomised to an experimental condition in which one of three vignettes was presented. The experimental vignettes stated the client was experiencing symptoms which were either "typical of…" either, "biological depression" or "psychosocial depression." In the third, control condition, a neutral vignette was presented in which the symptoms were simply framed as "typical of depression." All other information about the client was the same across conditions. The study examined clinician's causal models of depression with respect to the client vignette presented.

The study aimed to explore a.) whether framing the depression as a biological or psychosocial would bias trainees causal beliefs, and b) whether causal beliefs impact treatment decisions and attitudes.

Questionnaires examined the likely causes of the depression, the treatments that were felt to be most effectiveness, perceived controllability of the depression and perceived stigma. In addition, the questionnaire examined therapist optimism towards psychological treatment of the client.

Background and objectives

There is lack of research examining the impact of causal models on clinicians' attitudes. Previous research has suggested that biological explanations of depression increase clinical psychologists' perceptions of the effectiveness of medical treatments whilst reducing the perceived efficacy of psychological therapy. Causal explanations have also been shown to influence professional attitudes to depression. Studies suggest that biological explanations reduce perceptions of self-efficacy and control over symptoms. The current study aimed to explore whether clinicians' causal models of a client's depression can be biased by aetiological labelling and in turn, whether clinicians' causal models impact clinical judgements and attitudes.

This is of importance as there is no evidence to support varying treatment by depression subtype (NICE, 2009).

Findings

Data was analysed using ANOVA. There was a small effect of the manipulation in the predicted direction; labelling the depression as biological increased causal attributions of biological factors and reduced ratings of stress and negative life events as likely causes. In turn, the

biological condition led to increased perceptions of the effectiveness of medical treatments. Labelling depression as psychosocial did not affect causal beliefs or perceptions of treatment effectiveness but did seem to have a small effect on increasing perceptions of stigma. There was no effect of condition on ratings of psychological therapy, perceptions of controllability of the depression or on optimism for psychological treatment.

Exploratory analysis was also conducted to examine the impact of participants' primary causal beliefs about the depression on attitudes (this was independent of experimental group). There were substantial effects of strongly endorsing biological causal beliefs; this led to increased judgements of effectiveness of medical treatments (large effect) and lower perceptions of client engagement in therapy (medium effect). The data also suggested that people who more strongly endorsed biological explanations perceived the client to be more risky (e.g. self-harm). Participants who did not hold strong causal models of the depression rated the depression to be less controllable. There was no effect of causal models on stigma or perceptions of psychology therapy as being effective.

What did I conclude?

Findings suggest that clinicians' casual models of a client's depression can bias clinical-judgements. There were limitations to the current study, such as multiple self-report measures, no measure of behavioural intentions, and the use of multiple comparisons. In addition, this study examined attitudes to a client vignette and may not generalise to real -life settings. However, clinical vignettes are widely used in research and do have validity; clinicians often receive written referral information as first source of information about a client.

The study offers preliminary support that holding biological causal models of depression can bias judgements of treatment effectiveness and client engagement. Leading trainees to more strongly endorse medical treatments such anti-depressants, even when they are unlikely to be indicated by the evidence-base or client data and to perceive clients to be less likely to engage fully in psychological therapy. The effect sizes were substantive and warrant further confirmatory studies.

The experimental findings also suggested that labelling depression as biological can bias clinicians towards endorsing biological causal beliefs and medical treatments. This was a small effect and less conclusive. Given the subtly of the manipulation used in the study the effect seems worthy of further investigation. More research is needed to see if these findings can be reliably replicated.

Confidentiality

All data and personal information will be stored securely within Canterbury Christ Church University premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by the principal researcher and research supervisors. After completion of the study, all data will be remain anonymous.

Thank you again for your time and participation with this research study. If you have any questions regarding the study please contact the principal researcher on the contact details below.

Trainee Clinical Psychologist
Salomon's Campus, Canterbury Christ Church University
Email
Supervised by:

If I want to make a complaint about the research.
Please direct any complaints to Prof Margie Callanan,
Department of Applied Psychology, Canterbury Christ Church University, Broomhill Rd., Tunbridge Wells, Kent TN3 0TG

K. Reliability and normality of scales

Table 9.

Normality and reliability analysis

	α	item's deleted	α before item deleted	Number of items on final scale	Skew	Kurtosis
Biological	.87	No	-	6	-.37	-.24
Psychosocial	.66	No	-	11	.23	-.57
Medical treatments	.57	No	-	3	.45	-.33
Psychological treatment*	.67	No	-	3	-.63	.78
Self-initiated treatment	.73	"managing alone"	.51	4	.54	1.5
Self-medication**	.79	"vitamins/herbal"	.53	2	1.4	.17
Control-Cure	.54	No	-	6	-.85	.75
Control	.62	No	-	3	-.81	.17
Self-efficacy	.92	No	-	24	.13	.17

Stigma	.93	No	-	25	-1.11	2.15
Authoritarian	.74	No	-	6	-1.12	1.65
Discrimination	.93	No	-	5	-.85	.66
Dependency	.76	No	-	2	-.65	-.06
Dangerousness	.72	No	-	5	.21	.24
Affectivity	.84	No	-	7	-1.34	2.89
Engagement	.56	No	-	3	-.07	-.21
Treatment Effectiveness	.55	No	-	2	-.59	.48
Severity**	.39	No	-	2	-.56	.17
Risk**	.43	No	-	2	.37	.11

*One extreme outlier removed **Log transformations applied

L. Correlations

Table 10.

Correlation matrix

		B	PS	M	P	SI	SM	SE	CC	S	E	Eg	R	S
SEB: Biological (B)	*r* *p* N	1.00 217												
SEB: Psychosocial (PS)	*r* *p* N	-.05 .51 217	1.00 217											
STE: Medical (M)	*r* *p* N	.46** .00 217	-.05 .46 217	1.00 217										
STE: Psychological (P)	*r* *p* N	.04 .52 216	.12 .09 216	.06 .42 216	1.00 216									
STE: Self-Initiated (SI)	*r* *p* N	.07 .33 217	.16* .02 217	.05 .48 217	.45** .00 216	1.00 217								
STE: Self-medication (SM)	*r* *p* N	.03 .63 217	.04 .57 217	.11 .11 217	-.17 .02 216	-.04 .61 217	1.00 217							
Self-efficacy (SE)	*r* *p* N	.08 .22 217	.41** .00 217	.12 .07 217	.33** .00 216	.43** .00 217	-.03 .65 217	1.00 217						

Control-Cure (CC)	*r* *p* N	.06 .42 217	.14* .04 217	.09 .21 217	.30** .00 216	.24** .00 217	-.20** .01 217	.35** .00 217	1.00 217				
Overall stigma (S)	*r* *p* N	.04 .60 217	.24* .00 217	.11 .11 217	.156* .02 216	.20** .01 217	.04 .59 217	.24** .00 217	.08 .25 217	1.0 0 217			
CAQ: Effectiveness (E)	*r* *p* N	-.17 .09 217	.13 .06 217	-.03 .67 217	.25** .00 216	.16* .02 217	-.16** .02 217	.29** .00 217	.43** .00 217	.15* .03 217	1.00 217		
CAQ: Engagement (Eg)	*r* *p* N	-.16* .02 217	.13* .05 217	-.13* .05 217	.10 .16 216	.07 .32 217	-.11 .12 217	.19** .01 217	.10 .15 217	.05 .43 217	.20** .01 217	1.00 217	
CAQ: Risk (R)	*r* *p* N	.17* .01 217	-.03 .70 217	.06 .37 217	.14* .04 216	-.19** .01 217	-.05 .46 217	-.01 .89 217	-.09 .20 217	.01 .89 217	-.02 .73 217	-.22** .01 217	1.00 217
CAQ: Severity (S)	*r* *p* N	.06 .36 217	.20** .00 217	.04 .55 217	-.03 .71 216	.03 .68 217	.02 .81 217	.18** .01 217	-.11 .10 217	.14* .04 217	-.03 ..68 217	-.04 .61 217	.50** .00 217

*Correlation significant at the .05 level (two-tailed) **Correlation significant at the .01 level (two-tailed)

M. Interaction analysis

Causal beliefs about mental illness. Participants who scored above the mean on either biological (n= 93) or psychosocial (n= 44) causal beliefs about mental illness were extracted for use in the analysis. A two-way (3x2), group x mental illness beliefs, ANOVA was conducted. There was a large effect of mental illness beliefs on biological causal ratings, F(1,131)= 25.09, p<.001, ηp^2= .16, P= 1.00. People who felt biological causes were important in mental illness were more likely to see the cause of the clients depression as biological, relative to participants who only strangely endorsed psychosocial causes as important in mental illness (mean difference= 2.45, std. error= 0.65). There was no main effect of mental illness beliefs on psychosocial causal ratings, F(1,131)= 3.17,p= .077, ηp^2= .02, P= 0.42.

There was no significant interaction effect on biological, F(2,131)= 1.13, p= .33, ηp^2<.02, P= 0.25, or psychosocial causal ratings of depression, $F(2$,131)= .38, p= .68, ηp^2<.01, P= 0.11.

Year of training. A two-way (3x3), group x year, ANOVA was conducted. There was a significant main effect of year of training on ratings of biological causes, F(2,208)=

4.09, p= .02, ηp^2= .04, P= .72. Participants in the first year of training had significantly higher ratings of biological causality than those in their third year (mean difference = .30, p<.01, std. error= 0.11). There was no significant effect of year on psychosocial ratings of causality, $F(2,208)$= 0.99, p= .37, ηp^2<.01, P= 0.22.

There was no significant interaction effect on biological, $F(4,208)$= 0.54, p= .71, ηp^2= .01, P= 0.18, or psychosocial causal ratings, $F(4,208)$= 0.84,p= .50, ηp^2= .02, P= 0.27.

Identifying with a theoretical orientation. A two-way ANOVA (3x2), group x theoretical orientation (Yes, N= 53; No, N= 119) was conducted. There was a significant main effect on psychosocial causal ratings F (1, 166) = 5.72, $p<.02$, ηp^2= .03, P= .67. People who stated they had a theoretical orientation gave higher ratings of psychosocial causes (mean= 3.33, std. error= .05) compared to those who did not identify with a theoretical orientation (mean= 3.17, standard error= .04). There was no main effect of identifying with a theoretical orientation on biological causal ratings $F(1,166)$ = 1.47 p= .27, ηp^2= <.001, P= 0.23.

There was no significant interaction effect on biological causes, $F(2,166)$= 0.20, p= .82,ηp^2<.01,P= .08, or on psychosocial causes, $F(2,166)$ = .58, p= .94,ηp^2<.01, P= 0.06.

Gender. A two-way (3x2), group x gender, ANOVA was conducted. There was no main effect of gender on biological causal ratings, $F(1,211)$= .2.24, p= .13, ηp^2= .01, P= .32, or psychosocial causal ratings, $F(1,211)$ = .07, p= .79, ηp^2<.01, P= .06.

There was no interaction effect on biological , $F(2,166)$= 1.79, p= .17, ηp^2<.02, P= .37, or psychosocial causal ratings, $F(2,166)$= 1.39, p= .25,ηp^2= .01, P= .29.

N. Stigma as across experimental groups

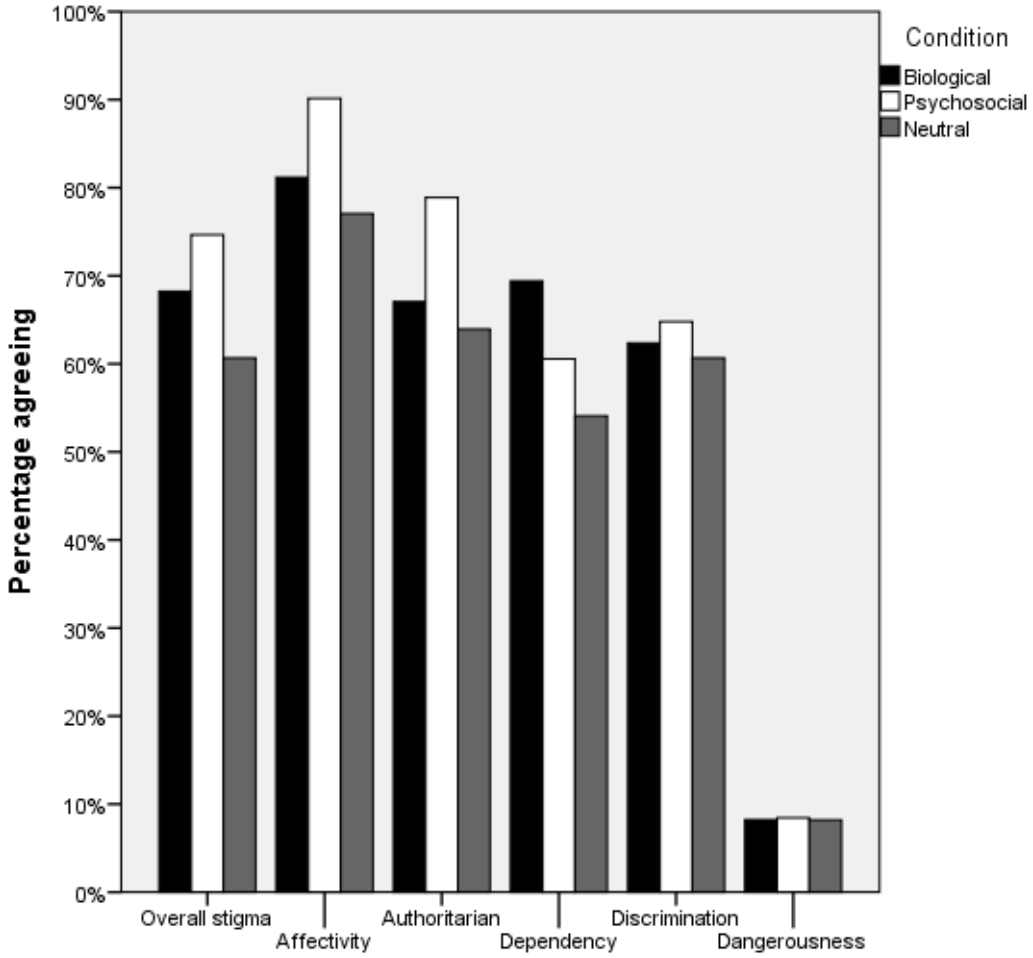


Figure 9. Perceived likelihood the client would experience stigma

O. Journal instructions

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